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ACUTE CORONARY SYNDROME

Clinical Outcomes in Patients with Acute Left Circumflex/Obtuse Marginal Occlusion Presenting with Myocardial Infarction

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Background: *Acute occlusion of left circumflex (LCx) or obtuse marginal (OM) arteries can present as ST elevation myocardial infarction (STEMI) or non-ST elevation myocardial infarction (NSTEMI). NSTEMI patients (pts) with occlusions have worse outcomes than those without occlusions, but no studies specifically examine outcomes in acute myocardial infarction (AMI) pts with LCx/OM occlusion. This study aims to define the incidence of NSTEMI in pts presenting with LCx/OM occlusion and analyzes clinical characteristics and outcomes in those presenting with NSTEMI compared to STEMI.*

Methods and Materials: *A review of our catheterization and STEMI database was performed to identify AMI pts presenting with LCx or OM occlusion from 1/1/2007 to 7/31/2009 at the Medical College of Virginia. Patients were divided into STEMI and NSTEMI groups, and a chart review was performed. Primary end-points were in-hospital mortality (HM), cardiogenic shock (CS), and in-hospital CHF. Secondary end-points included peak CK-MB and time to catheterization, as well as combined end-points of 1-month mortality, and recurrent AMI and CHF.*

Results: *Fifty-six pts met inclusion criteria, 54% of whom presented with NSTEMI. STEMI pts were significantly more likely to meet the primary end-points, as well as the combined secondary end-points. They had shorter times to catheterization but larger infarct sizes. Patients with left or mixed coronary dominance were more likely to have STEMI.*

Conclusions: *AMI pts with LCx/OM occlusion present with NSTEMI as often as STEMI. Those with NSTEMI have better outcomes, which may be related to right coronary dominance.*

Summary: *Patients with acute LCx or OM occlusion present with NSTEMI as often as STEMI, but those with STEMI have worse outcomes. The difference in presentation may be related to coronary dominance.* (J Interven Cardiol 2011;24:27–33)

Introduction

ST-segment elevation myocardial infarctions (STEMI) involving acute occlusion of the left circumflex (LCx) artery make up only 14–21% of STEMIs.[1] In an analysis of 5 STEMI trials, Krishnaswamy et al. found that only 14% of STEMIs resulted from LCx occlusion and noted that, in studies of non-ST-segment elevation acute coronary syndromes (NSTEACS), the distribution of coronary vessels is equal.[2] While clinical outcomes of STEMI involving the right coronary artery (RCA) and left anterior descending artery (LAD) are well studied, less is known about acute myocardial infarction (AMI) involving the LCx and obtuse marginal (OM) arteries. It is possible that acute plaque ruptures leading to occlusion occur less often in the LCx/OM, or these occlusions may be underrepresented in STEMI trials due to difficulty in diagnosing these occlusions with an electrocardiogram (ECG). In contrast to STEMIs secondary to acute occlusions of the RCA or LAD in which ECGs tend to easily identify occlusion by ST-segment elevations and/or Q waves, acute occlusion of the LCx is more difficult to acutely identify. Because of this difficulty, several studies have advocated additional

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ECG leads or other invasive/noninvasive diagnostic modalities to improve diagnosis of AMI secondary to vessel occlusion, particularly in the LCx system territory. Unfortunately, the utility of these methods has limitations.[2–6]

The lack of diagnostic ECG findings for acute occlusion of the LCx/OM system brings to light several important clinical questions, which our study tries to answer. This study aims to determine the true incidence of acute occlusions in the LCx/OM that present with non-ST-segment elevation myocardial infarction (NSTEMI) and analyze the difference in clinical outcomes in patients (pts) with acute LCx/OM occlusion who present with NSTEMI compared to STEMI. There are two potential hypotheses for outcomes in these pts. Conventional knowledge argues that pts presenting with NSTEMI may have better outcomes in the acute setting because their infarcts are not transmural. Alternatively, NSTEMIs in this setting may be "STEMI-equivalents," in which case the pts presenting with STEMI would have better or equal outcomes because of more urgent reperfusion than NSTEMI pts. The purpose of this study is to determine which of these hypotheses is true.

Materials and Methods

We used an IRB exemption to review the catheterization database, STEMI program database, and medical records at Medical College of Virginia to identify all pts who presented from January 2007 to July 2009 with AMI (defined as elevation in Troponin I and clinical symptoms consistent with AMI) thought to be secondary to acute LCx or OM occlusion on coronary angiography. The catheterization reports were used to define location of lesions, vessels involved, vessel dominance, presence of occlusion, and TIMI flow grade. If there was any ambiguity in the reports, the films were reviewed independently. An occlusion was defined as 100% angiographic occlusion, or TIMI flow grade 0–1 in the vessel with the lesion in question. The occlusion was classified as acute if it was the initial vessel intervened upon or if it was intervened upon on the initial catheterization procedure along with another vessel, presumably indicating that the interventional cardiologist felt the occlusion was not chronic and was the direct cause of or significant contributor to the AMI. Patients with prior coronary artery bypass graft surgery were excluded.

A retrospective chart review was performed to include clinical variables and outcomes, including history and physicals, discharge summaries, laband study results, and recurrent admission records or outpatient visits, if available. The pts were then separated into two groups: pts who had $\geq$1 mm ST-segment elevation in the anterior, inferior, and/or lateral leads, ST-depression in anterior leads with predominant R waves in leads V1 and V2 (findings consistent with posterior AMI), or new left bundle branch block (LBBB) were classified as STEMI; all other pts, including those with old LBBB, nonspecific ST-T wave changes, arrhythmias not definitively associated with myocardial injury, and normal ECGs were classified as NSTEMI. Primary end-points were all cause in-hospital mortality (HM), cardiogenic shock (CS) presumed to be secondary to the AMI, and in-hospital CHF. Secondary end-points included the combined end-point of mortality, recurrent AMI, and recurrent CHF in pts who had at least 1 month of follow-up available. If follow-up was available longer than 1 month, these outcomes were also included. Individual end-points or clinical characteristics that were not clearly ascertained from review of the medical record were not included in analysis for that particular category.

Baseline clinical characteristics, peak CK-MB levels, door to time to catheterization, ECG findings, and vessel characteristics were also included in the analysis. Peak CK and CK-MB values were used to determine infarct size. Peak TnI was not used, as the assay changed during the course of the study and results were not comparable. Time to catheterization for STEMI was obtained from our institution's NCDR door-to-balloon time log. In cases of STEMI or NSTEMI pts for which this was not available or other documentation of catheterization time was not stated, the time from initial ECG to first image in catheterization laboratory was used. For pts who were transferred from an outside facility in whom the initial ECG times or time of presentation to that hospital was not available, the time from presentation to our hospital until first angiographic image was used. In cases of transfer when our medical records indicated a time period of presentation to the outside hospital, the shortest possible time that the patient could have presented to our lab was used as catheterization time. The ECGs were reviewed by a cardiologist and confirmed by a second interpreter. If ECGs were unavailable, reported results from the chart were used for classification. Parametric Student's t-tests and chi-square tests were used for

Table 1. Patient Demographics

Clinical Characteristics	NSTEMI (N = 30) (%)	STEMI (N = 26) (%)	P Value
Age (years)	53.7	59.0	0.134
Male	25/30 (83.3)	16/26 (61.5)	0.066
White	18/30 (60.0)	10/25 (40.0)	0.140
DM	10/30 (33.3)	7/25 (28.0)	0.670
Hypertension	21/30 (70.0)	19/24 (79.2)	0.445
Hyperlipidemia	18/30 (60.0)	15/24 (62.5)	0.852
Smoking	20/28 (71.4)	15/22 (68.2)	0.804
Family history CAD	10/25 (40.0)	7/19 (36.8)	0.831
History CVA	3/29 (10.3)	3/25 (12.0)	1.000
History MI/PCI	15/30 (50.0)	4/25 (16.0)	0.011*
History PVD	4/29 (13.8)	2/25 (8.0)	0.675
Cocaine use	3/30 (10.0)	1/26 (3.9)	0.615
EF < 50%	11/27 (40.7)	8/25 (32.0)	0.513

*Statistically significant.
CAD = coronary artery disease; CVA = cerebrovascular accident; PCI = percutaneous coronary intervention; PVD = peripheral vascular disease; EF = ejection fraction; MI = myocardial infarction; CHF = congestive heart failure.

analysis of data using the JMP v.8.0 statistical software. A 2-tailed P value of ≤0.05 was considered significant.

Results

A total of 56 pts met inclusion criteria for the time period noted: 30 (54%) had NSTEMI and 26 (46%) had STEMI. Patient demographics are shown in Table 1. Baseline patient characteristics between groups were similar, except for a significantly higher percentage of pts with history of myocardial infarction (MI) or PCI in the NSTEMI group (P = 0.011).

Pts presenting with STEMI had significantly higher in-hospital all cause mortality (P = 0.017), in-hospital CHF (P = 0.008), and CS (P = 0.019) compared to those pts who had NSTEMI secondary to LCx/OM occlusion (Table 2). Secondary end-point analyses reveal a significantly higher combined end-point of mortality, recurrent MI, and CHF at ≥1-month follow-up in the STEMI group compared to the NSTEMI group (P = 0.029), although they did not reach statistical significance individually (Table 2). STEMI pts had larger infarcts with a significantly higher mean peak CK-MB of 264.6 ± 193 compared to a mean peak CK-MB of 87.6 ± 82.1 in NSTEMI pts (P < 0.001).

NSTEMI pts underwent coronary angiography later than STEMI pts with a mean time of at least 1,223 ± 934 minutes compared to 154 minutes ± 199 minutes, respectively (P < 0.001). This average delay to the catheterization lab in STEMI pts is likely attributed to multiple factors, most of which can be explained by delay in outside hospital transfer. Table 3 analyzes STEMI pts who had delay to catheterization, including reason for delay, whether they had thrombolytics, and primary end-points for each individual. In some cases, there were complicating factors that may have delayed presentation to catheterization lab. For example, patient #10 initially refused catheterization and required psychiatry consultation and patient #9 presented with cardiac arrest requiring resuscitation and stabilization. Patient #7 was an extreme example of delay in transfer who did not go to catheterization lab for

Table 2. Studies Examining ECG Sensitivity for Detecting Occlusion in MI by Artery

Study	Population	Artery	n = STE	P Value	Comments
Schmitt et al.	Analysis of 418 patients with MI and angiographic occlusion of a coronary artery, comparing ECG sensitivity for each artery	LCx RCA LAD	55 (46%) 117 (85%) 137 (85%)	NA	Adapted from Table 1 using standard ECG criteria of >2 mm STE
Berry et al.	Analysis of surface ECGs in cath lab while transiently occluding coronary arteries during PTCA	LCx RCA LAD	19 (32%) 12 (92%) 25 (84%)	<0.01	P value applies to LCx compared to LAD and RCA, individually
Huey et al.	Prospective study of 241 pts with CK-MB confirmed MI and artery occlusion to compare ECG changes by artery involvement	LCx RCA LAD	19 (48%) 76 (71%) 68 (72%)	0.012	38% w/LCx occlusion had no ST changes; 21% w/RCA occlusion and 20% with LAD occlusion had no ST changes (P = 0.001)

ECG = electrocardiogram; MI = myocardial infarction; STE = ST segment elevation; LCx = left circumflex artery; RCA = right coronary artery; LAD = left anterior descending artery; NA = not available; mm = millimeter; CK-MB = creatine kinase-MB.

Table 3. Analysis of STEMI Patients Who Had Delay to Catheterization

Patient	Reason for Delay	Thrombolytics?	In-hospital Mortality?	In-hospital CHF?	Cardiogenic Shock?
#1	Transfer	Yes—failed	No	Yes	Yes
#2	Transfer	No	No	No	No
#3	Transfer	No	No	No	No
#4	Unknown	No	Yes	Yes	Yes
#5	Transfer	No	Yes	Yes	Yes
#6	Unknown	No	No	No	No
#7	Transfer	No	No	No	No
#8	Transfer	Yes—failed	No	No	No
#9	Cardiac arrest, unclear diagnosis	Yes but given for PE	No	Yes	Yes
#10	Refused catheterization, psychiatry consultation	No	No	No	No
#11*	Transfer	Yes—failed	No	No	No

*Patients transferred from our hospital to another hospital prior to ultimate discharge.
Cath = catheterization; PE = pulmonary embolus.
End-points are from our hospital.

1,020 minutes. A comparison of the STEMI pts who had delay to catheterization and no delay to catheterization was made, but no statistical analysis was done due to small sample size. Of the 11 STEMI pts with delay to catheterization, 4 met primary end-points of in-hospital CS (36.4%) and CHF (36%), while 2 died in the hospital (18%). By comparison, of the 15 pts who did not have delay in catheterization, 3 had CS (21%), 4 had in-hospital CHF (29%), and 3 died in the hospital (20%). Those pts who had reperfusion with thrombolytics did not meet inclusion criteria since they were unlikely to have culprit occlusion of the LCx system on angiography. Five STEMI pts in our analysis received and failed thrombolytic therapy. Analysis of these 5 patients revealed that one had in-hospital mortality (20%), and 3 had in-hospital CHF and CS (60%).

The vessel and lesion characteristics by AMI type are listed in Table 4. Of the pts with left or mixed coronary dominance, 67% had STEMI, while only 37% of pts with right coronary dominance had STEMI. In other words, pts with a right dominant system were significantly less likely to have STEMI ($P = 0.037$). Of the 30 pts who had NSTEMI, the culprit lesion was equally distributed among the marginal and circumflex vessels. Of the 26 STEMI pts, 18 (69%) had circumflex involvement and 8 (31%) had marginal involvement ($P = 0.145$). Similarly, proximal lesions did not predict type of AMI as an equal proportion of NSTEMI pts had proximal lesions and mid/distal lesions, while 73% of STEMI pts had mid/distal lesions and 27% had proximal lesions ($P = 0.078$).

ECG findings in the NSTEMI pts are reported in Table 5. Nonspecific changes or normal ECGs made up the majority of ECGs in this group. It is unknown whether any of the ECG changes in NSTEMI pts are predictive of circumflex or marginal occlusion because in this study, there is no comparison made between pts who had NSTEMI secondary to nonocclusive lesions in the vessels in question. In the STEMI group, only 1 patient presented with a new LBBB (Table 6). The most common location of STEMI by ECG was in the inferior

Table 4. Vessel and Lesion Characteristics of Patients with NSTEMI Compared to STEMI

Vessel/Lesion Characteristics	NSTEMI (N = 30) (%)	STEMI (N = 26) (%)	P Value
Coronary dominance			
Right	24 (80.0)	14 (53.9)	0.037*
Left/mixed	6 (20.0)	12 (46.2)	
Vessel involvement			
Circumflex	15 (50.0)	18 (69.2)	0.145
Marginal	15 (50.0)	8 (30.8)	
Lesion location			
Proximal	15 (50.0)	7 (26.9)	0.078
Mid/distal	15 (50.0)	19 (73.1)	

*Statistically significant.

Table 5. Characteristic ECG Changes in Patients with NSTEMI (n = 27)*

ECG Finding	%
Inferior ST depression	1 (3.7)
Anterior ST depression	6 (22.2)
Lateral ST depression	9 (33.3)
Nonspecific/no changes	17 (63.0)

*Some patients had more than one ECG finding.

and/or lateral territories, which were often accompanied by ST segment changes in the anterior leads.

Discussion

The results of this retrospective study support conventional knowledge, clearly showing that those pts with LCx/OM occlusion presenting with STEMI had larger infarcts and worse short- and long-term outcomes as compared to NSTEMI pts with similar angiographic findings. Patients presenting with NSTEMI may have better outcomes than pts who present with STEMI due to nontransmural infarcts and less myocardium at risk. However, multiple studies have suggested that this may not necessarily be true in all circumstances, particularly in infarcts involving the posterior/lateral territories. Wang et al. determined that NSTEMI secondary to acute occlusion conferred larger infarcts and higher risk-adjusted 6-month mortality similar to that of STEMI pts, terming these cases "STEMI equivalents."[4] Dixon et al. also found that NSTEMI secondary to occluded culprit vessel led to higher rates of in-hospital mortality, CS, and CHF compared to NSTEMI without occlusion.[5] In a subanalysis of the Triton-TIMI 38 study presented at American Heart Association conference in 2008 examining pts who presented with isolated anterior ST-segment depressions on ECG, many were initially misdiagnosed as NSTEMI, leading to a significant delay to percutaneous coronary intervention, higher recurrent AMI rate with larger infarcts, and increased 30-day mortality. A review by Krishnaswamy et al. analyzed potential consequences of unidentified acute occlusions of the LCx by citing the aforementioned studies by Wang et al. and Gibson et al.[2] This study also notes that a disproportionate number of NSTEACS pts with LCx occlusion and large infarcts enrolled in the FRISC-II trial had worse 12-month outcomes[7] and that pts who had AMI due to LCx occlusion benefited equally from early reperfusion whether they had ST-segment elevations on ECG or not.[8] Our study is the first to specifically analyze the LCx/OM system and include STEMI pts, clarifying the apparent contradiction between conventional knowledge and the aforementioned studies, and demonstrating that LCx/OM occlusion in the setting of NSTEMI confers less risk than STEMI.

Table 6. Characteristic ECG Changes in Patients with STEMI (n = 24)*

ECG Finding	%
New LBBB	1 (4.2)
Inferior ST elevation	17 (70.1)
Anterior ST elevation	0 (0.0)
Lateral ST elevation	11 (45.8)
Changes consistent with posterior MI	10 (41.7)

*Some patients had more than one ECG finding.
LBBB = left bundle branch block.

Discrepancy in Infarct Size for STEMI versus NSTEMI in Patients with LCx/OM Occlusion

It is unclear why our NSTEMI pts with LCx/OM occlusion had smaller infarcts than STEMI pts with similar occlusions. There are several potential explanations, some of which relate to myocardium at risk. It is well known that the LCx supplies less myocardium than the LAD and if nondominant, the RCA as well. In a pathologic study of fatal infarcts, size of infarcts involving the RCA and LCx occupied respective means of 18% and 20% of the left ventricular myocardium, compared to a mean of 40% of the left ventricular myocardium in infarcts involving the LAD. Furthermore, the size of infarct was directly related to ischemic bed size.[9] Obviously, this study did not include a potentially large number of survivors who had AMI due to occlusion of a LCx/OM that represented a small ischemic bed size. However, the O'Keefe study revealed that pts with LCx occlusion and NSTEMI had the same amount of myocardium involved in their infarcts as those with STEMI and RCA or LCx occlusion.[8] Furthermore, in a study analyzing AMI pts with nonischemic ECGs, Kontos et al. determined that the most common infarct related artery was the LCx, and infarcts involving this vessel were larger by CK-MB compared to LAD, with similar involvement of myocardium compared to other vessels.[10] These data would seem to argue against the

theory of less myocardium at risk as an explanation for smaller infarct size, suggesting myocardium at risk may not be the only contributing factor to infarct size.

It seems likely that right coronary dominance may provide a protective effect for acute occlusions of the LCx/OM system, minimizing infarct size. Our data revealed smaller infarct size and higher likelihood of right coronary dominance in NSTEMI pts, which would support this explanation.

Discrepancy in Primary and Secondary End-points for STEMI versus NSTEMI in Patients with LCx/OM System Occlusion

As expected, pts in our study with STEMI went to the catheterization laboratory much faster than those with NSTEMI. However, the expectation that this more aggressive management strategy with earlier reperfusion would lead to better outcomes was not consistent with our results. In our study, angiographic findings do not appear to be as predictive of outcome as the ECG itself. The lack of characteristic ST-segment elevation on ECG correlated somewhat with vessels that may have supplied less myocardium and were nondominant. Therefore, while it may be true that NSTEMI pts with occluded coronaries have improved outcomes compared to those without occlusions,[4,5] we take exception to the term "STEMI equivalent." Our findings clearly show that pts who have NSTEMI secondary to occlusion have smaller infarcts and better outcomes than STEMI pts, at least when isolating the LCx/OM system.

One potential explanation for this discrepancy is that many of the aforementioned studies analyzed all vessels with occlusions. Inclusion of pts with acute occlusions of the RCA and LAD who have NSTEMI selects pts who are less likely to have nondiagnostic ECGs in AMI (due to much higher sensitivity for ECG to detect acute occlusions) and selects vessels that are usually larger, supplying more myocardium, often without dual blood supply. In addition, it also increases the likelihood of including pts with chronic occlusions. While it is possible that occlusions of RCA or LAD without ST-segment elevations are equivalent to STEMI of these territories, the term "STEMI equivalent" may not apply to pts with NSTEMI and LCx/OM occlusion. O'Keefe et al. proposes that "if patients with acute circumflex infarction without ST-segment elevation can be identified and reperfused, they will benefit as much as those with LCx or right coronary infarction with ST-segment elevation."[8] This statement was based upon myocardial salvage rather than clinical outcomes, and all NSTEMI pts underwent urgent revascularization within 6 hours, which is not standard of care. There was no comparison group of NSTEMI pts with LCx occlusion who did not have urgent revascularization and the lower-risk NSTEMI pts were not included.

Electrocardiogram in Predicting LCx/OM Occlusion in AMI

It has been well documented in the literature that ST-segment elevation on a standard 12-lead ECG is not sensitive for diagnosing acute occlusions of the LCx. While a standard 12-lead ECG is able to diagnose acute occlusion of the LAD and RCA with 85–90% and 70–90% sensitivity, respectively, it is only able to diagnose occlusion of the LCx with 32–50% sensitivity.[3,6,11–13] Similarly, over half of our pts with AMI due to LCx/OM occlusion presented without characteristic ECG changes suggestive of STEMI. We believe that pts with LCx/OM system occlusion, compared to occlusion of other major coronary vessels, may have a tendency to present more frequently with NSTEMI because of the vessel's posterior location, placing it farthest from the chest wall and ECG leads, and making it difficult to detect on 12-lead ECG.[4] The lack of sensitivity for EKG detection of significant infarcts of the LCx is supported by a study comparing pts with RCA occlusion to LCx occlusion, revealing those with LCx occlusion had larger infarcts and more left ventricular dysfunction, despite having lesser magnitude of ST-segment elevation.[14] While additional ECG leads can be helpful in diagnosing infarcts occurring in the LCx system territory, they are not routinely used or foolproof, and still leave many acute infarcts undiagnosed.[6,15,16] Our data seem to suggest that the location of the LCx/OM system relative to ECG leads is not the only factor contributing to its poor sensitivity in detecting acute occlusions.

The ability of the ECG to predict LCx/OM occlusion in NSTEMI pts could be accomplished by comparing ECG findings of NSTEMI pts presenting with LCx/OM lesions. However, the utility of such an exercise is yet to be determined. This evaluation would be useful only if the clinical outcomes in pts with NSTEMI and LCx/OM occlusion were determined to be worse in those pts who had occlusion.

Study Limitations

There are several limitations of this study. It has all of the disadvantages of a retrospective analysis. Although data were available for the primary end-points, a proportion of pts lacked follow-up data, thus compromising the secondary end-point analysis. Our study assumed that an occluded vessel occurred acutely if intervened upon in the initial catheterization by the interventional cardiologist. Inclusion of more pts would have been ideal, but we did not extend our analysis prior to 2007 because computerized medical records for this time period were limited. Furthermore, we wanted to allow for the most contemporary treatments that are most pertinent today.

The mean time to coronary angiography for STEMI pts was 154 minutes, which is much longer than expected, as the mean door-to-balloon time for all STEMI pts presenting to our emergency department over a similar time period was 61 minutes. The time delay in our pts is attributed to a relatively large percentage of transfers and some complicated cases that delayed presentation to the lab. Time to catheterization for certain STEMI pts (particularly transfers) was inconsistent as some were not logged in our NCDR door-to-balloon time log and had to be obtained by other potentially unreliable methods. Time to catheterization in the NSTEMI group was underestimated due to the fact that presentation times to outside hospitals were not well documented, leading us to use the minimal time possible for the patient to present to the catheterization lab.

Conclusion

Prior studies give clear evidence that NSTEMI pts with acute occlusion have worse clinical outcomes than NSTEMI pts without occlusion. Our study suggests that acute occlusions of LCx/OM system presenting with NSTEMI have much better outcomes than those presenting with STEMI, indicating that all LCx/OM occlusions are not created equally. The reason for difficulty in diagnosing LCx/OM occlusion by ECG may not be related to the posterior location of the vessel as much as it is related to the frequency with which this vessel supplies a small amount of myocardium and is nondominant. Our study indicates that there is a better relationship to the ECG and prognosis than there is to LCx/OM occlusion, despite a generally more aggressive management strategy in the group with larger infarcts.

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