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ORIGINAL ARTICLE

Angiographic and Clinical Predictors of Non-Culprit Coronary Lesion Progression after Percutaneous Coronary Intervention in Patients with ST-Elevation Myocardial Infarction.

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ABSTRACT

Background: Most of cardiovascular (CV) events in patients of ST-elevation myocardial infarction (STEMI) with undergoing primary percutaneous coronary intervention (PPCI) are linked to the progression of non-culprit coronary lesions (NCCLs) during the follow-up period. Yet, the clinical and angiographic risk factors of non-culprit coronary lesion (NCCL) progression are not well known.

So this study was aimed to assess the clinical risk profile and angiographic features that can be related to the progression of mild NCCLs (<50% stenosis) of patients with STEMI and undergoing PCI over 24 months duration.

Methods: The present cohort study evaluated 200 patients with STEMI underwent PCI to the culprit lesion and have mild, non-culprit lesion (NCL) <50% stenosis. Patients were divided into 2 groups; Non progressed group: 157 patients with non-progressed NCLs. Progressed group: 43 patients who needed additional PCI to progressed NCLs.

Results: In a multivariate logistic regression analysis, independent predictors of NCLs progression included the age (odds ratio [OR]:0.854, 95% confidence interval [CI]: 0.775 to 0.941; p=0.001), C-reactive protein (CRP) (OR:1.670, 95% CI: 1.255 to 2.223; p < 0.001), non-targeted low density lipoprotein cholesterol (LDL-C) with follow up (>1.4mmol/L) (OR:14.030, 95% CI: 2.766 to 71.155; p= 0.001), Complex culprit lesion (OR:47.249, 95% CI: 4.925 to 453.290; p = 0.001), and presence of more than one NCL (OR:27.090, 95% CI: 4.213 to 174.179; p= 0.001).

Conclusions: The underlying clinical and angiographic characteristics can predict NCLs progression. Complex morphology of culprit lesion is the strongest independent predictor for progression of NCLs.

Keywords: *STEMI, culprit, PCI, NCCL, Clinical predictors.*

INTRODUCTION

The primary percutaneous coronary intervention (PPCI) is the reperfusion procedure of choice in patients with ST-elevation myocardial infarction (STEMI) with the availability of skilled operators in high-volume centers, surgical back-up, and within 90 minutes of the first medical contact with the patients [1].

Several randomized trials and meta-analysis have shown that early and routine post-thrombolysis angiography (within 3 to 24 hours of administration) with subsequent percutaneous coronary intervention (PCI) will decrease the levels of recurrent ischemia and re-infarction compared to the watchful waiting protocol in which angiography and revascularization were maintained only in patients with left ventricular dysfunction or re-infarction [2].

Patients with acute myocardial infarction (AMI) and have non-culprit coronary lesion (NCCL) of complex morphology are at higher risk of repeated intervention after successful PCI [3].

It was found that the event of acute STEMI can be clinically considered as an independent predictor for the progression of NCCL. The peak count of circulating monocytes and C-reactive protein (CRP) during hospitalization might predict the progression of coronary plaque [4].

The vulnerable plaques can exist at NCCL, exactly as in the culprit site in patients with acute coronary syndrome (ACS) [5]. Moreover, patients with ACS who underwent PCI had the same rate of recurrent

adverse cardiac event in both culprit and non-culprit lesions (NCLs). So, detection of these non-obstructive and vulnerable plaques has an important role in the prevention of AMI and sudden cardiac death [6].

The moderate to severe stages of chronic kidney disease (CKD) can play an important role in the progression of the NCCL after culprit lesion PCI [7].

We aimed to identify patients with STEMI who are at higher risk for mild NCCL progression after PCI (either PPCI or pharmaco-invasive PCI).

METHODS

Patient's population This cohort study conducted on 200 patients, who were admitted to the coronary care unit with STEMI and underwent PPCI done in 144 patients and pharmaco-invasive PCI done in 56 patients to the culprit artery and have mild non culprit lesions (< 50% stenosis) were enrolled in the study and followed up over 24 months. Patients were divided into 2 groups (based on whether the clinically driven mild non culprit lesion PCI existed or not); Non progressed group formed of 157 patients with non-progressed non culprit lesion and progressed group formed of 43 patients with clinically driven non culprit lesion PCI.

The study protocol was formally reviewed and approved by the ethics committee for human research at Zagazig Faculty of Medicine with informed consent obtained from all participants prior to commencement of the study after a thorough explanation of the study objectives. The study was carried out in accordance with recommendations of the Declaration of Helsinki.

Time frame 30 months from 1st of April 2017 to 1st of October 2019.

Inclusion Criteria Acute STEMI (i.e., evidence of ischemic chest pain for > 30 minutes and new ST-segment elevation in two or more contiguous electrocardiographic (ECG) leads and elevated cardiac troponin), the culprit artery which shows a de novo lesion in a native vessel of ≥ 2.5mm in diameter with TIMI (Thrombolysis in Myocardial Infarction) flow grade from 0 to 2 and mild NCCL that shows a stenosis of less than 50%.

Exclusion Criteria Killip class ≥ 3, Left Bundle Branch Block (LBBB), Infarcted Related Artery (IRA) with excessive proximal tortuosity or severe calcification, Left Ventricular Ejection Fraction (LVEF) < 40%, lack of clinical follow up, hospital death after PCI, Myocardial Infarction (MI) within 2 weeks of PCI to exclude potential subacute stent thrombosis of the intervened up on artery, repeated PCI of culprit coronary lesions for restenosis and patients who had a non-culprit lesion >50 % but, still insignificant and did not need PCI as judged by the operators.

Methods: All patients were subjected to detailed history taking including coronary artery disease (CAD) risk factors, physical examination and ECG to detect ST segment and T wave abnormalities.

Cardiac biomarkers, fasting blood glucose, glycated hemoglobin (HbA1c), lipid profile, CRP, serum creatinine and creatinine clearance were measured.

Conventional Echocardiography measure of LVEF (measured from apical two and four chamber views using modified Simpson's biplane method) and wall motion abnormalities using the Philips Echo machine and the results were done blindly by two echo experts as recommended by the American Society of Echocardiography.

Mild lesion was defined in our study as that non culprit lesion of less than 50% stenosis seen during the index PCI for the culprit lesion. All patients were followed for 24 months after the index PCI to report whether this mild lesion will progress or not.

Uncontrolled DM was defined as failure to achieve the average of HbA1c < 7-8% during follow up period. HbA1c < 7% was the target to judge good controlled DM in most of our diabetic patients, however we applied the less stringent target (HbA1c < 8%) in some patients who have a long duration of diabetes, multiple comorbidities or ages ≥ 70 [8].

Uncontrolled HTN was defined as an average SBP ≥ 140 mm Hg or an average DBP ≥ 90 mm Hg among those with hypertension [9] during the follow up duration.

Uncontrolled low density lipoprotein cholesterol (LDL-C) was defined as the average of LDL-C failed to achieve the recommended LDL-C target (<1.4mmol/L or < 55mg/dL) [10] during the follow up duration.

The coronary lesions were classified in our study according to the American College of Cardiology/American Heart Association classification (type A, B1, B2, and C) based on the morphological characteristics of lesions that cause stenosis of the coronary arteries. We then categorized the lesions into two categories according to the mentioned classification: simple lesions (A or B1 lesions) and complex lesions (B2 or C). Type A: Discrete (<10mm), non-angulated segment < 45°, smooth contour, little or no calcification, less than totally occlusive, not ostial in location, no major branch involvement and absence of thrombus. Type B1: lesion that has one of the following characteristics: tubular (10-20mm), eccentric, moderate tortuosity of proximal segment, moderately angulated (45-90°), irregular contour, moderate to heavy calcification, ostial in location or bifurcation lesions requiring double guidewire and some thrombus present. Type B2: lesion that has two or more of the above

characteristics mentioned in type B1 lesion. Type C: diffuse (> 20mm length), excessive tortuosity of proximal segment, extremely angulated (>90°), inability to protect major side branch and degenerated vein graft with friable lesions [11].

Primary PCI: Emergent PCI with balloon, stent or other approved device performed in the IRA without previous fibrinolytic treatment [12].

Rescue PCI: Emergent PCI performed as soon as possible in case of failed fibrinolytic treatment (<50% ST-segment resolution at 60-90 min or at any time in the presence hemodynamic or electrical instability or worsened ischemia) [12].

Routine early PCI strategy: Coronary angiography with PCI of the IRA if indicated that is performed between 2 and 24 hours after successful fibrinolysis (ST-segment resolution > 50% in 60–90min, typical reperfusion arrhythmia and the disappearance of chest pain) [12].

Coronary angiography was performed using the percutaneous radial approach by Sildenger technique. Right and left coronary angiography was performed using multiple projections and analysis was done by professional interventionists who were blind to clinical data.

During the index procedure all patients underwent PCI to the culprit lesion and any apparently significant (as judged by the operators) non culprit lesion (NCL), either on the same setting or on another setting before hospital discharge.

Statistical analysis:

Data were coded and entered using the statistical package for the Social Sciences (SPSS) version 26 (IBM Corp., Armonk, NY, USA). Data was summarized using mean, standard deviation, median, minimum and maximum in quantitative data and using frequency (count) and relative frequency (percentage) for categorical data. Comparisons between quantitative variables were done using the non-parametric Mann-Whitney tests. For comparing categorical data, Chi square ($\chi 2$) test was performed. Exact test was used instead when the expected frequency is less than 5. Logistic regression was done to detect independent predictors of NCL progression. P-values less than 0.05 were considered as statistically significant.

RESULTS

Current smoker, dyslipidemia, positive family history of CAD, depression, CKD stage III or more ($p < 0.001$ for each), OSA, history of old CVA ($p = 0.009$ for each), auto-immune disease ($p = 0.002$) and previous MI or PCI ($p = 0.032$ for each), were found to have a significant increase in the progressed group. Gender distribution showed insignificant difference between both groups with male patients represented in 90.7% in the progressed group versus 92.36% in the non-progressed group. After following up clinical characteristics, it was found that uncontrolled DM and/or HTN showed significant increase in the progressed group than non-progressed group, where uncontrolled HTN represented in 18 patients (41.86%) in the progressed group versus 11 patients (7.01%) in non-progressed group ($p < 0.001$) and uncontrolled DM represented in 21 patients (48.84%) in the progressed group versus 10 patients (6.37%) in non-progressed group ($p < 0.001$). Thirty-four patients (79.07%) in the progressed group did not achieve the LDL-C target versus only 16 patients (10.19%) in the non-progressed group ($p < 0.001$) (Table 1).

Regarding age, the mean age of the progressed group 52.05 ± 9.64 versus 63.31 ± 9.16 in the non-progressed ($p<0.001$). The CRP and peak cardiac troponin I values showed significant elevations in patients of the progressed group in comparison to the values reported in the non-progressed group, where the mean value of CRP was 10.32±4.48mg/L in the progressed group versus 4.69±2.52mg/L in the non-progressed group ($p < 0.001$) and peak cardiac troponin I showed a mean value of 44.37±20.47µ/L in the progressed group versus 27.10±13.44µ/L in non-progressed group (Table 2).

Complex culprit lesion, more than one NCL, thrombotic culprit lesion, culprit long lesion > 20mm, and ≥ 2 vessels lesion showed significant increase among our patients of the progressed group ($p < 0.001$ for each) represented in 28 patients (65.1%) in the progressed group versus 23 patients (14.6%) in non-progressed group , 29 patients (67.4%) in the progressed group versus 14 patients (8.9%) in non-progressed group, 27 patients (62.79%) in the progressed group versus 23 patients (14.65%) in non-progressed group, 25 patients (58.14%) in the progressed group versus 10 patients (6.37%) in non-progressed group and 20 patients (46.51%) in the progressed group versus 14 patients in non-progressed group (8.92%) respectively (Table 3).

Regarding the morphological characteristics of NCL in the progressed and non-progressed lesions, Types B2 and C lesions (complex lesions) showed a significant increase in the progressed NCLs ($p < 0.001$ for each), where they represented 52.83% and 13.21% respectively in the progressed lesions versus 11.89% and 1.08% respectively in non-progressed lesions. While type B1 lesion showed insignificant difference between both groups, where it represented 30.19% in the progressed lesions versus 38.92% in the non-progressed lesions ($p = 0.246$). Type A lesion was found to be significantly increased in the non-progressed NCLs, where it represented 3.77% in the

progressed lesions versus 48.11% in the non-progressed lesions (p < 0.001) (Table 4).

Forty-three patients (53 NCLs) received additional PCI in NCLs for NCLs progression, whereas the mean time to progression was 20.98 ± 6.30 months with a range between 7-30 months and the degree of stenosis of these NCLs was 82.2 ± 10.72 % at the time of additional PCI, while the degree of stenosis was 34.89 ± 6.68 % in the setting of STEMI (Table 5).

In a multivariate logistic regression analysis, independent predictors of NCLs progression included the age (odds ratio [OR]:0.854, 95% confidence interval [CI]: 0.775 to 0.941; p=0.001), CRP (OR:1.670, 95% CI: 1.255 to 2.223; p < 0.001), non-targeted LDL-C with follow up(>1.4mmol/L) (OR:14.030, 95% CI: 2.766 to 71.155; p= 0.001), Complex culprit lesion (OR:47.249, 95% CI: 4.925 to 453.290; p = 0.001) and the presence of more than one NCL (OR:27.090, 95% CI: 4.213 to 174.179; p= 0.001), however elevated CRP was the most significant predictor (Table 6).

Table (1): Baseline and follow up clinical characteristics in patients of progressed and non-progressed groups:

		Progressed group (n=43)		Non progressed group (n=157)		P value
		Count	%	Count	%	
Current smoker		36	83.72%	52	33.12%	< 0.001
Sex	**M**	39	90.70%	145	92.36%	0.752
	F	4	9.30%	12	7.64%	
DM	**yes**	24	55.81%	69	43.95%	0.167
	no	19	44.19%	88	56.05%	
HTN	**yes**	23	53.49%	75	47.77%	0.506
	no	20	46.51%	82	52.23%	
PAD	**yes**	1	2.33%	1	0.64%	0.385
	no	42	97.67%	156	99.36%	
Controlled DM with follow up (HbA1c < 7-8%)	**no**	21	48.84%	10	6.37%	< 0.001
	yes	3	6.98%	58	36.94%	
Controlled BP with follow up (< 140/90mmHg)	**no**	18	41.86%	11	7.01%	< 0.001
	yes	5	11.63%	63	40.13%	
LDL-C not targeted with follow up(>1.4mmol/L)	**yes**	34	79.07%	16	10.19%	< 0.001
	no	9	20.93%	141	89.81%	
Dyslipidemia		21	48.84%	32	20.38%	< 0.001
CKD (≥ stage III)		7	16.28%	0	0.00%	< 0.001
Family history		16	37.21%	5	3.18%	< 0.001
Obstructive sleep apnea		3	6.98%	0	0.00%	0.009
Auto-immune disease		4	9.30%	0	0.00%	0.002
Depression		6	13.95%	1	0.64%	< 0.001
Previous MI		3	6.98%	1	0.64%	0.032
Previous PCI		3	6.98%	1	0.64%	0.032
Old CVA		3	6.98%	0	0.00%	0.009

BP= blood pressure, DM= diabetes mellitus, HTN= hypertension, PAD= peripheral artery disease, LDL-C= low density cholesterol lipoprotein, CKD= chronic kidney disease, MI= myocardial infarction, PCI= percutaneous coronary intervention, CVA= cerebrovascular insult, HbA1c= glycated hemoglobin.

Table (2): Age, CRP, and peak cardiac troponin I value in relation to patients of progressed and non-progressed groups:

	Progressed group (n=43)					Non progressed group (n=157)					P value
	Mean	SD	Median	Minimum	Maximum	Mean	SD	Median	Minimum	Maximum	
Age	52.05	9.64	52.00	33.00	76.00	63.31	9.16	65.00	32.00	82.00	< 0.001
Peak troponin I(µ/L)	44.37	20.47	40.00	18.00	110.00	27.10	13.44	23.00	7.00	70.00	< 0.001
CRP (mg/L)	10.32	4.48	11.00	2.50	17.00	4.69	2.52	4.00	1.00	14.00	< 0.001

CRP= C-reactive protein, SD= standard deviation, NCL= non culprit lesion

Table (3): The baseline angiographic characteristics in patients of progressed and non-progressed groups:

	progressed group (n=43)		Non progressed group (n=157		P value
	Count	%	Count	%	
≥2 Vessel lesions	20	46.51%	14	8.92%	< 0.001
Culprit lesion length >20mm	25	58.14%	10	6.37%	< 0.001
Thrombotic culprit lesion	27	62.79%	23	14.65%	< 0.001
Complex culprit lesion	28	65.1%	23	14.6%	< 0.001
>1 NCL	29	67.44%	14	8.92%	< 0.001
MBG (0-1)	14	32.56%	54	34.39%	0.822
Pre-dilatation	42	97.67%	143	91.08%	0.200

NCL= non culprit lesion, MBG= myocardial blush grade.

Table (4): Baseline morphological characteristics of non-culprit lesions in relation to progressed and non-progressed lesions:

		NCL				
		Progressed lesions(n=53)		Non-progressed lesions(n=185)		P value
		Count	%	Count	%	
Type A lesion	+	2	3.77%	89	48.11%	<0.001
	-	51	96.23%	96	51.89%	
Type B1 lesion	+	16	30.19%	72	38.92%	0.246
	-	37	69.81%	113	61.08%	
Type B2 lesion	+	28	52.83%	22	11.89%	<0.001
	-	25	47.17%	163	88.11%	
Type C lesion	+	7	13.21%	2	1.08%	<0.001
	-	46	86.79%	183	98.92%	

Type B2 or C are classified as complex lesions, while type A or B1 are classified as simple lesions. Type A= lesion that is: discrete (<10mm), non-angulated segment <45°, smooth contour, little or no calcification, not ostial in location, no major branch involvement. Type B1= lesion with one of the following: tubular(10-20mm), moderate tortuosity of proximal segment, moderately angulated 45-90°, irregular contour, moderate to heavy calcification, ostial in location, bifurcation lesion. Type B2= having two or more of characteristics mentioned in type B1. Type C= lesion that is: diffuse (>20mm length), excessive tortuosity of proximal segment, extremely angulated >90°, inability to protect major side branch, NCLs= non culprit lesions, NCL= Non culprit Lesion

Table (5): Percentage of progressed NCLs driven PCI, and median time of its progression:

	Patients No= 200	NCLs No.= 238		Went for NCL driven PCI		Time till progression (months)
		Progressed	Non-progressed	Patients No.	NCLs No.	Mean ±SD: 20.98 ± 6.30
Non progressed	157(78.5%)	0(0.0%)	171(71.8%)	0	0	
progressed	43(21.5%)	53 (22.3%)	14 (5.9%)	43	53	Range: 7 – 30

SD= Standard Deviation, NCL= non culprit Lesion, PCI= percutaneous coronary intervention, No= number

Table (6): Multivariate logistic regression analysis to detect independent predictors of NCL progression:

		P value	OR	95% C.I.	
				Lower	Upper
NCL progression	**Age**	0.001	0.854	0.775	0.941
	CRP (mg/L)	<0.001	1.670	1.255	2.223
	LDL-C not targeted with follow up(>1.4mmol/L)	0.001	14.030	2.766	71.155
	Complex culprit lesion	0.001	47.249	4.925	453.290
	>1 NCL	0.001	27.090	4.213	174.179

NCL= non culprit lesion, CRP= C - reactive protein, LDL-C= low density lipoprotein cholesterol, OR= odds ratio, C. I= confidence interval

DISCUSSION

It is known that ACS patients are at high risk for recurrent ischemic events, which are caused by a lesion that is anatomically unrelated to the initial event" [13].

In this study, we investigated clinical and angiographic factors that may predict the progression of NCLs. Clinical follow-up was performed in all patients. There were 238 NCLs in 200 patients in the setting of STEMI and 43 patients (21.5%) (53 NCLs progressed to culprit lesions) underwent clinically driven NCLs PCI. There were no de novo lesions that progressed to culprit lesions. A total of 157 patients (78.5%) (171 NCLs did not progress to culprit lesion) did not undergo additional PCI (non-progressed group). This indicated that NCL progression may be the key cause of revascularization after PCI for patients with STEMI.

In the current study there were significant differences in age, where patients with progression of NCL were significantly younger and most often males, this came in agreement with Li J et al. [14] in addition, they reported that STEMI has significantly occurred in young patients than in older ones and this high AMI prevalence would play a role in the NCLs rapid progression in young patients.

In the current study there were no significant differences in gender and incidence of DM and HTN between the two groups. Meanwhile, there were significant differences in smoking, dyslipidemia, CKD and family history of CAD between the two groups with significant increase in the progressed group patients.

In contrast to our results, Wang et al. [15] and Sanidas et al. [16] found that age, hyperlipidemia and current smoking were not significantly different in between both groups. This disparity may be explained by varying risk factors among different populations.

In the current study there were significant differences in the previous history of MI and PCI between the two groups. This came in disagreement with Wang et al. [15], Sanidas et al. [16] and Brener et al. [17] who found no significant difference in previous MI and PCI in between both groups.

In the current study the marker of inflammation CRP was reported significantly with higher values in the progressed group than that in the non-progressed group suggesting that inflammation may play a crucial role in NCL progression. This data came in line with Wang et al. [15], who found that CRP was significantly higher in the progression group than non-progression group. On the other hand, Data shown by Sanidas et al. [16] and Brener et al. [17] was inconsistent to our study as it found that there was no significant difference between the two groups regarding CRP.

With follow up clinical characteristics, the current study found that poorly targeted LDL-C, uncontrolled DM and uncontrolled HTN were reported significantly in the progressed group compared to the non-progressed group. These data were supported by Shin et al. [18], Kataoka et al. [19] and Bayturan et al. [20] regarding the effect of LDL-C, uncontrolled DM, and uncontrolled HTN respectively, suggesting that these clinical factors can participate in NCL progression.

Comparing between progression group and non-progression group, the current study showed that there was a significantly higher thrombotic culprit lesion rate, complex culprit lesion rate, ≥2 vessel lesions rate and > one NCL rate. These results indicate that these characteristics may influence the progression of NCLs and predispose to an additional PCI. This came completely consistent with Wang et al. [15] who found that thrombotic lesion rate, complex lesion rate and ≥2 vessel lesion rate were significantly higher in the progression group than that in the non-progression group.

Many studies supported the same conclusions. Kataoka et al. [19] noticed that multiple complex lesions were reported with a higher percentage in the progressed group than the non-progressed group. Kaski et al. [21] suggested that in patients with stable angina complex lesions are associated with rapid CAD progression. Park et al. [22] found that in the progressed group (NCL- PCI group), the percentage of significant multivessel CAD was significantly higher in the progressed group than that in the non-progressed group.

In the current study there was a significant difference in the length of culprit lesion between the two groups that was longer in the progressed group than that in the non-progressed group, suggesting that the extent of the culprit lesion with its underlying burden can affect the progression of NCLs. This was agreed with Wang et al. [15] who had found the same results of significantly longer culprit lesion in the progressed co-group than that in non-progressed group.

In the current study, baseline morphological characteristics of NCLs revealed that the rate for lesions, type B2 and C were significantly higher in the progressed NCLs than NCLs that did not progress to culprit lesions ($P<0.001$ for each). This came in to balance with Wang et al. [15],

In multivariate logistic regression analysis, the current study showed independent predictors of clinically driven PCI for the progression of NCLs included the age, CRP, non-targeted LDL-C with

follow up, Complex culprit lesion and the presence of more than one NCL, where CRP is the most significant predictor. So, we are raising a red flag for the elevated admission CRP and also why the younger patients are more susceptible for coronary lesion progression? Is it due to poorly controlled life style only? Are they having an underlying extensive inflammatory response when compared to older patients? We think that issue may need more concern.

Nakachi et al. [23] reported that in a multivariate analysis, admission CRP elevation, post-PCI CRP elevation and multiple complex lesions were independent predictors of rapid progression of NCLs.

Kang et al. [24] reported that independent predictors of non-culprit-ischemia driven revascularization were diabetes and lesion type B2/C.

Our study had some limitations, the criteria for inclusion included only acute STEMI and The follow-up in our study was only 2 years, where the prognostic implications of mild lesion progression may change after this interval.

CONCLUSIONS

This study suggests that Mild NCLs seen at the index PCI for STEMI patients can progress, whereas the underlying clinical and angiographic characteristics can predict NCLs progression. Younger age, complex morphology of culprit lesion, poorly targeted LDL-C, admission CRP and presence of more than one NCL are independent predictors of progression of NCLs. The present study suggests that among these predictors admission CRP is the strongest independent predictor for the progression of NCLs.

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