

# Impact of Chronic Thrombocytopenia on In-Hospital Outcomes After Percutaneous Coronary Intervention



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## ABSTRACT

**OBJECTIVES** This study sought to evaluate the impact of chronic thrombocytopenia (cTCP) on clinical outcomes after percutaneous coronary intervention (PCI).

**BACKGROUND** The impact of cTCP on clinical outcomes after PCI is not well described. Results from single-center observational studies and subgroup analysis of randomized trials have been conflicting and these patients are either excluded or under-represented in randomized controlled trials.

**METHODS** Using the 2012 to 2014 National (Nationwide) Inpatient Sample database, the study identified patients who underwent PCI with or without cTCP as a chronic condition variable indicator. Propensity score matching was performed using logistic regression to control for differences in baseline characteristics. The primary outcome of interest was in-hospital mortality. Secondary outcomes of interest included in-hospital post-PCI bleeding events, post-PCI blood and platelet transfusion, vascular complications, ischemic cerebrovascular accidents (CVAs), hemorrhagic CVAs, and length of stay.

**RESULTS** Propensity matching yielded a cohort of 65,130 patients (32,565 with and without cTCP). Compared with those without cTCP, PCI in patients with cTCP was associated with higher risk for bleeding complications (odds ratio [OR]: 2.40; 95% confidence interval [CI]: 2.05 to 2.72;  $p < 0.0001$ ), requiring blood transfusion (OR: 2.10; 95% CI: 1.80 to 2.24;  $p < 0.0001$ ), requiring platelet transfusion (OR: 11.70; 95% CI: 6.00 to 22.60;  $p < 0.0001$ ), higher risk for vascular complications (OR: 1.94; 95% CI: 1.43 to 2.63;  $p < 0.0001$ ), ischemic CVA (OR: 1.60; 95% CI: 1.20 to 2.10;  $p = 0.01$ ), and higher in-hospital mortality (OR: 2.30; 95% CI: 1.90 to 2.70;  $p < 0.0001$ ), but without a significant difference in hemorrhagic CVA (OR: 1.50; 95% CI: 0.70 to 3.10;  $p = 0.27$ ).

**CONCLUSIONS** In this large contemporary cohort, patients with cTCP were at higher risk of a multitude of complications, including higher risk of in-hospital mortality. (J Am Coll Cardiol Intv 2018;11:1862-8)

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Platelets play a significant role in the pathogenesis of acute coronary syndrome (ACS) (1,2). Treatment for ACS involves platelet inhibition using antiplatelet agents, anticoagulant therapy, and often, percutaneous coronary intervention (PCI) (3,4). Dual antiplatelet therapy is associated with an increased risk of bleeding (5), and this risk is higher

in those with thrombocytopenia (TCP) (6). On the other hand, presence of TCP is not protective against ACS (7,8). Although the exact mechanism is unknown, several hypothesis such as increased production of young, hyper-reactive platelets, toxicity of drugs used to treat the cause of TCP and coexisting antiphospholipid antibodies have been described (9). The

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incidence of TCP in the general population may be as high as 4% and is increasing due to an aging population (10). PCI in patients with TCP can be challenging due to not only the platelet abnormality but also the increased number of comorbidities that may modulate the risk (11). Data on the safety of PCI in this population is scant, and contradictory results have been reported from single-center observational studies and pooled secondary analysis of randomized controlled trials (11-13). To help understand the safety of PCI in this understudied population, we hypothesized that patients with chronic thrombocytopenia (cTCP) experience more early complications after PCI than do those without cTCP and designed the current study to test this in a large inpatient database.

SEE PAGE 1869

## METHODS

**DATA SOURCES.** The National (Nationwide) Inpatient Sample (NIS) is a publicly available, all-payer inpatient database maintained by the Agency for Healthcare Research and Quality. Annually, the NIS is composed of discharge-level data from roughly 8 million hospitalizations and approximates a stratified sample of 20% of community hospitals in the United States. The sampling methodology of the NIS permits the application of weighting variables that allow for the calculation of national estimates, which have been validated against other U.S. hospital registries (14). Each hospitalization within the database contains clinical and resource-use information, including but not limited to, age, sex, race, insurance status, primary and secondary procedures, hospitalization outcome, total cost, and length of stay. Patients' diagnoses are documented in parallel, as both International Classification of Diseases-9th Edition-Clinical Modification (ICD-9-CM) and clinically meaningful clusters of ICD-9-CM codes, termed Clinical Classification Software codes.

**STUDY SELECTION, ENDPOINTS, AND DEFINITIONS.** We searched for hospital admissions between 2012 and 2014 with a primary or secondary diagnosis of TCP, during which PCI was performed (ICD-9-CM codes 36.06 and 36.07). TCP is generally defined as platelets count of <150,000 cells/ $\mu$ l of blood. In the NIS, this diagnosis is based on inclusion as an ICD code and the exact lab values are not available. To identify cTCP, we used a chronic condition indicator variable, which identifies a diagnosis that was present for at least a year from the index admission. Patients with periprocedural TCP, or for whom the chronic condition indicator variable indicated that cTCP was

present for <1 year before procedure, were excluded, along with those with thrombotic thrombocytopenic purpura, those with disseminated intravascular coagulation, and those who underwent coronary artery bypass during index admission. The ICD-9-CM codes used to identify each of these diagnoses and procedures are listed in [Online Table 1](#). Common in-hospital complications of PCI such as post-procedural hemorrhage, post-procedural red blood cell (RBC) and platelet transfusions, vascular complications (injury to blood vessel, arteriovenous fistula, retroperitoneal hematoma, and vascular complications requiring surgical intervention), cardiac tamponade requiring pericardiocentesis, post-procedural acute cerebrovascular accident, in-hospital mortality, and length of stay were identified. These complications were identified by corresponding ICD-9-CM diagnosis and procedure codes ([Online Table 2](#)).

**STATISTICAL ANALYSIS.** We used the weights provided with the NIS to generate national estimates of the number of admissions during each year. Chi-square test was used to compare categorical variables and Wilcoxon signed rank sum test to compare continuous variables. Propensity matching was performed with the use of a nonparsimonious multivariable logistic regression model with cTCP as the dependent variable and all the baseline characteristics outlined in [Table 1](#) as covariates. Matching was performed with the use of a 1:1 matching protocol with a caliper width equal to 0.20 of the SD of the logit of the propensity score. Standardized differences were estimated for all the baseline covariates before and after matching. Differences in outcomes were reported as adjusted odds ratios (ORs) with 95% confidence intervals (CIs). Multivariable logistic regression was performed to identify higher risk subgroups among those with cTCP. Variables included age, sex, and statistically significant variables derived from the univariate analysis as well as other variables that may plausibly be associated with mortality. All statistical analyses incorporated primary sampling units and clusters to obtain national estimates and were performed using SAS version 9.4 (SAS Institute, Cary, North Carolina) with a 2-sided significance level set at  $p < 0.05$ .

## RESULTS

**BASELINE CHARACTERISTICS AND MATCHED COHORT.** Between 2012 and 2014, there were 1,435,215 PCIs ([Figure 1](#)). After excluding patients with

## ABBREVIATIONS AND ACRONYMS

**ACS** = acute coronary syndrome(s)  
**cTCP** = chronic thrombocytopenia  
**CI** = confidence interval  
**CVA** = cerebrovascular accident  
**ICD-9-CM** = International Classification of Diseases-9th Edition-Clinical Modification  
**NIS** = National (Nationwide) Inpatient Sample  
**OR** = odds ratio  
**PCI** = percutaneous coronary intervention  
**RBC** = red blood cell  
**TCP** = thrombocytopenia

<b>TABLE 1 Baseline Characteristics</b>						
	<b>Unmatched Cohort</b>			<b>Matched Cohort</b>		
	<b>With cTCP (n = 35,180)</b>	<b>Without cTCP (n = 1,395,825)</b>	<b>p Value</b>	<b>With cTCP (n = 32,565)</b>	<b>Without cTCP (n = 32,565)</b>	<b>p Value</b>
Mean age, yrs	68.8	64.4	<0.0001	68.8	69.3	0.49
Female	9,400 (26.7)	457,260 (32.8)	<0.0001	8,770 (26.9)	8,495 (26.1)	0.28
Hypertension	27,110 (77.1)	1,071,835 (76.8)	0.61	25,095 (77.1)	25,370 (77.9)	0.25
Race			0.02			0.11
White	25,240 (71.7)	1,004,365 (72.0)		25,005 (76.8)	25,705 (78.9)	
African American	2,705 (7.7)	118,115 (8.5)		2,660 (8.2)	2,470 (7.6)	
Hispanic	2,475 (7.0)	97,885 (7.0)		2,445 (7.5)	2,205 (6.8)	
Diabetes mellitus	16,080 (45.7)	552,185 (39.6)	<0.0001	14,940 (45.9)	15,260 (46.9)	0.27
Chronic kidney disease	10,650 (30.3)	206,250 (14.8)	<0.0001	9,920 (30.5)	10,345 (31.8)	0.12
Coronary artery disease	33,140 (94.0)	1,320,540 (94.6)	0.16	30,700 (94.3)	30,810 (94.6)	0.42
Congestive heart failure	13,675 (38.9)	287,005 (20.6)	<0.001	12,665 (38.9)	12,920 (39.7)	0.37
Atrial fibrillation	7,805 (22.2)	163,700 (11.7)	<0.0001	7,210 (22.1)	7,060 (21.7)	0.55
Prior cerebrovascular accident	3,040 (8.6)	97,015 (7.0)	<0.0001	2,875 (8.8)	2,445 (7.5)	0.05
Peripheral vascular disease	6,030 (17.1)	154,860 (11.1)	<0.0001	5,585 (17.2)	5,525 (17.0)	0.78
Valvular heart disease	6,090 (17.3)	136,290 (9.8)	<0.0001	5,620 (17.3)	5,505 (16.9)	0.60
Chronic liver disease	2,105 (6.0)	16,210 (1.1)	<0.0001	1,975 (6.1)	1,580 (4.9)	0.05
Prior percutaneous coronary intervention	6,875 (19.5)	289,745 (20.8)	0.16	6,375 (19.6)	6,255 (19.2)	0.59
Prior coronary artery bypass surgery	2,915 (8.3)	95,425 (6.8)	<0.0001	2,725 (8.4)	2,535 (7.8)	0.22
Prior pacemaker implantation	1,415 (4.0)	31,930 (2.3)	<0.0001	1,315 (4.0)	1,315 (4.0)	1.00
Prior ICD implantation	1,190 (3.4)	25,085 (1.8)	<0.0001	1,115 (3.4)	925 (2.8)	0.06
ST-segment elevation myocardial infarction	10,120 (28.7)	405,310 (29.0)	0.64	9,315 (28.6)	9,470 (29.1)	0.56
Non-ST-segment elevation myocardial infarction	13,940 (39.6)	481,300 (34.5)	<0.0001	12,815 (39.4)	12,610 (38.7)	0.47
Bare-metal stents	11,615 (33.0)	297,240 (21.2)	<0.0001	9,900 (30.4)	7,132 (21.9)	<0.0001
Drug-eluting stents	23,565 (67.0)	1,098,585 (78.8)	<0.0001	22,670 (69.6)	25,420 (78.1)	<0.0001
Hospital location and teaching status			<0.0001			0.97
Rural	2,185 (6.2)	85,935 (6.2)		1,970 (6.1)	2,000 (6.1)	
Urban nonteaching	10,950 (31.1)	479,075 (34.3)		10,525 (32.3)	10,475 (32.2)	
Urban teaching	22,045 (62.7)	830,815 (59.5)		20,070 (61.6)	20,090 (61.7)	

Values are n (%), unless otherwise indicated.  
ICD = implantable cardioverter-defibrillator; cTCP = chronic thrombocytopenia.

acute TCP, periprocedural TCP, disseminated intravascular coagulation, thrombotic thrombocytopenic purpura, or those who underwent coronary artery bypass grafting in the index admission, 1,431,005 patients were identified and included in the study. cTCP was present in 35,180 patients (2.5%). When compared with those without cTCP in the overall cohort, patients with cTCP were older and experienced more comorbidities, such as diabetes mellitus, congestive heart failure, atrial fibrillation, cerebrovascular accident, peripheral vascular disease, and valvular heart disease (Table 1). Propensity matching identified a sample of 65,130 patients (32,565 in each group) with well-matched baseline characteristics (Table 1).

**OUTCOMES.** In patients with cTCP, bare-metal stents were more commonly used (30.4% vs. 21.9%;  $p < 0.0001$ ) whereas drug-eluting stent use was lower

(69.6% vs. 78.1%;  $p < 0.0001$ ). Compared with those without cTCP, PCI in patients with cTCP was associated with an increased risk of post-procedure hemorrhage (OR: 2.40; 95% CI: 2.05 to 2.72;  $p < 0.0001$ ) as well as increased use of RBC (OR: 2.10; 95% CI: 1.80 to 2.24;  $p < 0.0001$ ) and platelet (OR: 11.70; 95% CI: 6.00 to 22.60;  $p < 0.0001$ ) transfusions (Table 2). There was a significantly increased number of vascular complications (OR: 1.94; 95% CI: 1.43 to 2.63;  $p < 0.0001$ ) as well as a higher risk of cardiac tamponade requiring pericardiocentesis (OR: 2.80; 95% CI: 1.40 to 5.40;  $p = 0.001$ ). The risk of post-procedural ischemic cerebrovascular accident (CVA) risk (OR: 1.60; 95% CI: 1.20 to 2.10;  $p = 0.01$ ) was higher, but the risk of hemorrhagic CVA (OR: 1.50; 95% CI: 0.70 to 3.10;  $p = 0.27$ ) was similar. Hospital stay was significantly longer in cTCP ( $6.90 \pm 0.25$  days vs.  $4.50 \pm 0.13$  days;  $p < 0.0001$ ). Finally, there was a >2-fold increase in

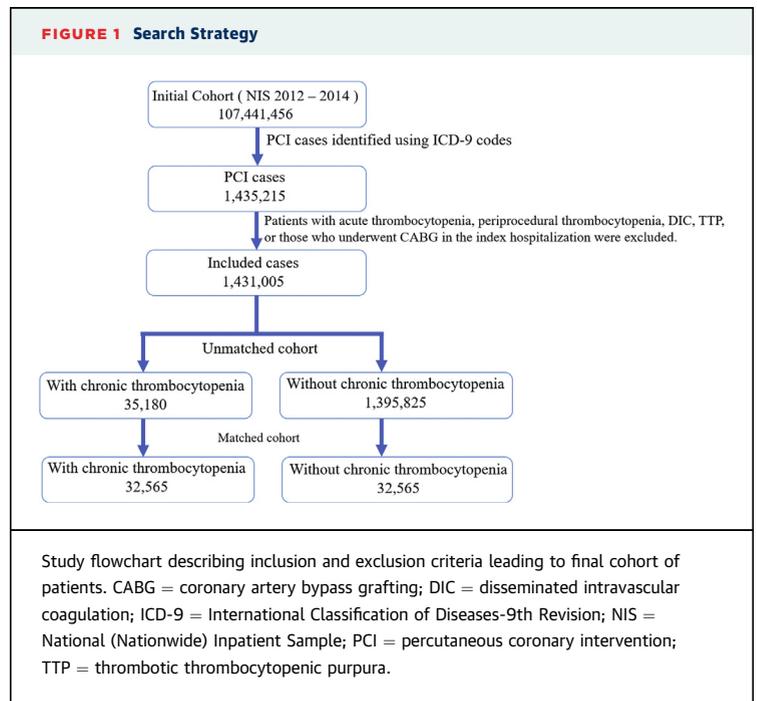
risk of in-hospital mortality (OR: 2.30; 95% CI: 1.90 to 2.70;  $p < 0.0001$ ) (Figure 2). A total of 14.7% of those who had post-operative bleeding requiring blood transfusion died, whereas post-operative bleeding requiring transfusion occurred in 21.5% of those who died. This suggested that bleeding complications alone do not explain the increased mortality in this population.

Subgroup analysis was performed to compare urban and rural hospitals (Table 3). When compared with patients treated in rural hospitals, patients treated in urban hospitals had higher odds of receiving RBC transfusion (OR: 1.58; 95% CI: 1.02 to 2.45;  $p = 0.037$ ) and higher in-hospital mortality (OR: 1.81; 95% CI: 1.09 to 3.00;  $p = 0.019$ ). Among urban hospitals, teaching hospital status was associated with lower odds of receiving RBC and platelet transfusions and a higher risk of hemorrhagic CVA but no difference in in-hospital mortality (OR: 1.10; 95% CI: 0.88 to 1.37;  $p = 0.42$ ).

We then investigated if RBC or platelet transfusion was associated with adverse outcomes. Patients with cTCP were stratified based on blood or platelet transfusion status (Table 4). In patients with cTCP, those who received RBC or platelet transfusion had a higher risk of acute ischemic CVA, hemorrhagic CVA, and in-hospital mortality.

To determine if transfusions were independent predictors of mortality in patients with cTCP, we performed multivariable logistic regression. Several variables were found to be predictors of mortality, including age (1-year increment, OR: 1.02; 95% CI: 1.01 to 1.03;  $p < 0.0001$ ), female sex (OR: 1.30; 95% CI: 1.07 to 1.55;  $p = 0.009$ ), congestive heart failure (OR: 1.70; 95% CI: 1.45 to 2.08;  $p < 0.0001$ ), atrial fibrillation (OR: 1.35; 95% CI: 1.10 to 1.60;  $p = 0.004$ ), ST-segment elevation myocardial infarction (OR: 6.10; 95% CI: 4.76 to 7.85;  $p < 0.0001$ ), non-ST-segment elevation myocardial infarction (OR: 2.22; 95% CI: 1.71 to 2.89;  $p < 0.0001$ ), peripheral vascular disease (OR: 1.45; 95% CI: 1.17 to 1.81;  $p = 0.0008$ ), post-procedural RBC transfusion (OR: 2.10; 95% CI: 1.63 to 2.70;  $p < 0.0001$ ), post-procedural platelet transfusion (OR: 3.64; 95% CI: 2.24 to 5.92;  $p < 0.0001$ ), acute ischemic CVA (OR: 2.39; 95% CI: 1.55 to 3.10;  $p < 0.0001$ ), and acute hemorrhagic stroke (OR: 13.11; 95% CI: 4.59 to 37.46;  $p < 0.0001$ ).

In view of the unexpected finding that ischemic CVA was more common after PCI in cTCP, we performed a multivariate analysis to identify risk factors. Female sex (OR: 1.53; 95% CI: 1.14 to 2.05;  $p = 0.005$ ), history of stroke (OR: 1.62; 95% CI: 1.06 to 2.45;  $p = 0.02$ ), congestive heart failure (OR: 1.69; 95% CI: 1.26 to 2.26;  $p = 0.0005$ ), atrial fibrillation (OR: 1.5;



95% CI: 1.10 to 2.04;  $p = 0.01$ ), ST-segment elevation myocardial infarction presentation (OR: 1.72; 95% CI: 1.28 to 2.32;  $p = 0.0003$ ), and peripheral vascular disease (OR: 1.58; 95% CI: 1.13 to 2.20;  $p = 0.007$ ) were associated with a higher risk of ischemic CVA in those with cTCP whereas RBC and platelet transfusions were not.

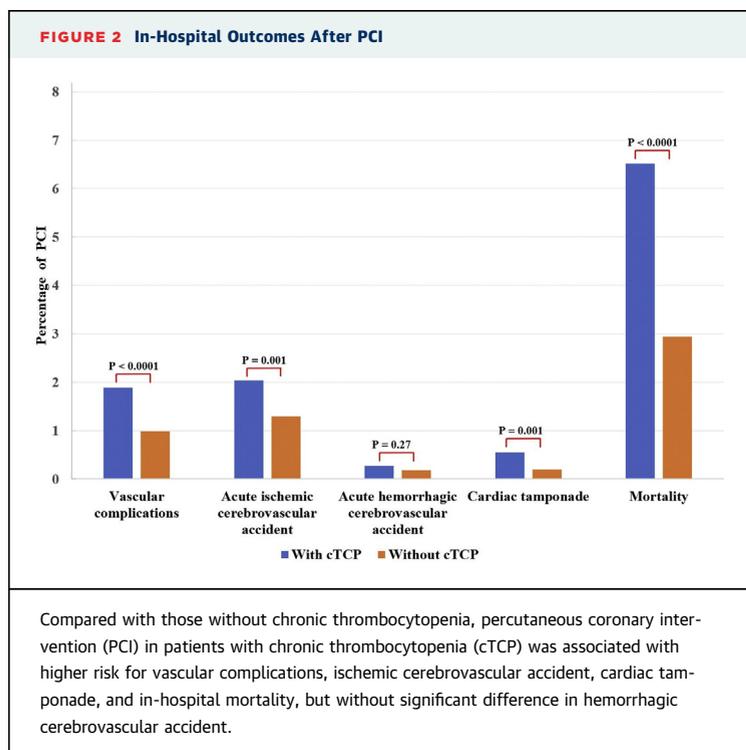
Finally, to determine if the etiology of cTCP was a predictor of outcomes, we studied differences in between 2 etiologies that could be easily identified using ICD codes—immune-mediated TCP and chronic liver disease. There were no significant differences in

**TABLE 2 Outcomes After PCI**

In-Hospital Outcomes	With cTCP (n = 32,565)	Without cTCP (n = 32,565)	Odds Ratio (95% CI)	p Value
Post-procedural hemorrhage	3,545 (10.9)	1,600 (4.9)	2.40 (2.05-2.72)	<0.0001
RBC transfusion	3,090 (9.5)	1,555 (4.8)	2.10 (1.80-2.24)	<0.0001
Platelet transfusion	630 (1.9)	55 (0.2)	11.70 (6.00-22.60)	<0.0001
Vascular complications	615 (1.9)	320 (1.0)	1.94 (1.43-2.63)	<0.0001
Acute ischemic CVA	664 (2.0)	420 (1.3)	1.60 (1.20-2.10)	0.001
Acute hemorrhagic CVA	89 (0.3)	60 (0.2)	1.50 (0.70-3.10)	0.27
Cardiac tamponade	179 (0.5)	65 (0.2)	2.80 (1.40-5.40)	0.001
Length of hospitalization, days	6.90 ± 0.25	4.50 ± 0.13		<0.0001
Mortality	2,120 (6.5)	960 (2.9)	2.30 (1.90-2.70)	<0.0001

Values are n (%) or mean ± SD, unless otherwise indicated.

CI = confidence interval; cTCP = chronic thrombocytopenia; CVA = cerebrovascular accident; PCI = percutaneous coronary intervention; RBC = red blood cell.



clinical outcomes in patients with these diagnoses compared with others with cTCP (Online Tables 3 and 4).

## DISCUSSION

PCI in a patient with an increased baseline risk of bleeding can be challenging. Of all bleeding disorders, TCP is the most common but these patients are typically excluded from clinical trials and single-center studies are underpowered to detect significant differences. The current analysis addresses this clinically relevant question in a large, propensity-matched cohort. First, the incidence of cTCP was as high, as 2.5% of all patients undergoing PCI. This is similar to the incidence in the general population and validates the case finding approach in the current study while underscoring the need to optimize outcomes in an increasing common disease. Second, PCI in cTCP was associated with a higher risk of bleeding complications, ischemic CVA, and mortality after PCI. Although the increased bleeding risk and hemorrhagic stroke is intuitive, the higher rate of ischemic CVA is puzzling. Because these patients were well matched in their baseline characteristics, differences in PCI strategies or post-PCI care might have played a role. Patients with cTCP might have received less intensive anticoagulation during PCI. Patients with cTCP also received a greater number of platelet and RBC

transfusions; these interventions may actually be proinflammatory and thrombogenic (15,16). Although administration of blood products (RBCs and platelets) after coronary artery bypass surgery appears to increase the risk of stroke in a dose-dependent manner, other studies of individual blood components have yielded conflicting results (17,18). However, transfusion was not a significant predictor of ischemic CVA in this analysis, suggesting that the risk was mediated by other factors.

Patients with TCP (except the mildest of cases) are often excluded from randomized controlled trials; single-center observational studies are often underpowered and may reflect institutional practice patterns. With these limitations, it is not surprising that prior studies in this area have provided conflicting data. In a single-center retrospective cohort analysis, patients with TCP undergoing PCI had higher in-hospital mortality (1.9% vs. 0.6%;  $p < 0.001$ ) and had higher in-hospital major bleeding events (1.7% vs. 0.8%;  $p < 0.05$ ) (12). This increased risk occurred despite a relatively mildly reduced mean platelet count ( $124,000 \pm 25,000/\text{mm}^3$ ). On the contrary, a retrospective analysis from the Mayo Clinic evaluated post-PCI outcomes in patients with platelet count  $<100,000/\text{mm}^3$  ( $n = 204$ ) (13). After propensity matching, patients with cTCP had similar rates of in-hospital bleeding events (16.4% vs. 14.0%;  $p = 0.4$ ) and in-hospital mortality (2.7% vs. 2.0%;  $p = 0.56$ ) compared with those with normal platelet counts. In this study, the median platelet count was  $85,500/\text{mm}^3$  (interquartile range: 72,500 to  $94,500/\text{mm}^3$ ), whereas only 12 patients had a platelet count  $<50,000/\text{mm}^3$ .

Yadav et al. (11) performed a pooled analysis of patients with cTCP included in 2 large randomized controlled trials: the ACUITY (Acute Catheterization and Urgent Intervention Triage Strategy) and HORIZONS-AMI (Harmonizing Outcomes With Revascularization and Stents in Acute Myocardial Infarction) trials. These trials studied the use of bivalirudin in patients with ACS and excluded patients with platelet count  $\leq 100,000/\text{mm}^3$ . In this analysis, patients with such relatively mild cTCP ( $n = 607$ ) were older and experienced more comorbidities when compared with those without cTCP. Thirty days post-PCI, there was no significant difference in the crude event rates in those with cTCP (all-cause mortality: 1.8% vs. 1.4% [ $p = 0.45$ ], MI: 5.8% vs. 4.9% [ $p = 0.30$ ], noncoronary artery bypass grafting major bleeding: 5.1% vs. 5.4% [ $p = 0.78$ ]).

The available data to date is thus inconclusive and indeed counterintuitive to what one would expect while performing invasive procedures in patients

**TABLE 3 Differences in Outcomes in Patients With cTCP Based on Urban or Rural Status and Urban Teaching or Nonteaching Status**

In-Hospital Outcomes	Urban Teaching (n = 20,070)	Urban Nonteaching (n = 10,525)	Odds Ratio (95% CI)	p Value	Urban (n = 30,595)	Rural (n = 1,970)	Odds Ratio (95% CI)	p Value
Post-procedural hemorrhage	2,260 (11.3)	1,100 (10.5)	1.09 (0.91-1.3)	0.36	3,360 (11.0)	185 (9.4)	1.19 (0.84-1.69)	0.33
Post-procedural RBC transfusion	1,730 (8.6)	1,235 (11.7)	0.71 (0.59-0.85)	0.0002	2,965 (9.7)	125 (6.3)	1.58 (1.02-2.45)	0.0368
Post-procedural platelet transfusion	325 (1.6)	275 (2.6)	0.61 (0.42-0.89)	0.0099	600 (2.0)	30 (1.5)	1.29 (0.58-2.88)	0.5281
Vascular complications	375 (1.9)	225 (2.1)	0.87 (0.59-1.29)	0.49	600 (2.0)	15 (0.8)	2.61 (0.60-11.38)	0.1853
Acute ischemic CVA	440 (2.2)	200 (1.9)	1.16 (0.78-1.72)	0.47	640 (2.1)	25 (1.3)	1.67 (0.67-4.10)	0.26
Acute hemorrhagic CVA	80 (0.4)	10 (0.1)	4.21 (0.97-18.3)	0.037	90 (0.3)	0 (0.0)		
Cardiac tamponade	130 (0.6)	45 (0.4)	1.52 (0.69-3.34)	0.29	175 (0.6)	5 (0.3)	2.26 (0.30-16.82)	0.41
Mortality	1,380 (6.9)	665 (6.3)	1.10 (0.88-1.37)	0.423	2,045 (6.7)	75 (3.8)	1.81 (1.09-3.00)	0.0193

Values are n (%), unless otherwise indicated.  
 Abbreviations as in Table 2.

with cTCP and may lull operators into a false sense of security. In the current study, which examined a large cohort over multiple centers and operators, there was an unequivocal increase in bleeding complications and mortality in those with cTCP when compared with those without in an otherwise well matched cohort. Importantly, the risk of mortality was 2 to 3 times higher than that reported in prior studies. The cause of mortality is unclear; in multivariate analysis, several clinical, ischemic, and hemorrhagic complications as well as transfusion were significant predictors. This suggests that as a group, these patients are less likely to tolerate both the disease process that led to PCI and the procedure itself.

These findings call for studies to improve outcomes in this population, especially in the setting of ACS. In the patient with ACS and cTCP, treatment strategy should be individualized (19). Techniques to reduce bleeding complications include use of radial access, micropuncture technique during femoral artery cannulation, and reduced dose of heparin, especially in those with severe TCP. In accordance with current guidelines and with the increased bleeding risk, PCI should be deferred in cTCP patients with stable coronary artery disease

unless significant symptoms persist despite maximal medical therapy.

**STUDY LIMITATIONS.** First, the NIS is an administrative database prepared by trained coding specialists after reviewing inpatient medical records to capture provider care and services that warrant reimbursement. Therefore, the data are subject to potential oversights in documentation and coding, just as in any other administrative database. We could not eliminate the effect of unmeasured confounders that might have contributed to the reporting of adverse effects. Second, we could not stratify the analysis based on severity of chronic TCP, because the mechanism of CAD and outcomes may be different between immune mediated and non-immune-mediated TCP. We studied only 2 etiologies of cTCP because clear delineation of etiology using ICD-9-CM codes can be challenging. Third, we only included patients with cTCP and the effect of shorter durations of TCP (<1 year) was not studied. Similarly, data on anticoagulation strategy, procedural technique, access site (radial vs. femoral), and medication use pre- and post-PCI was not available. Despite these limitations, the large number of

**TABLE 4 Patients With cTCP Stratified Based on Blood and Platelet Transfusion Status**

In-Hospital Outcomes	RBC Transfusion (n = 3,090)	No RBC Transfusion (n = 29,475)	Odds Ratio (95% CI)	p Value	Platelet Transfusion (n = 630)	No Platelet Transfusion (n = 31,935)	Odds Ratio (95% CI)	p Value
Acute ischemic CVA	130 (4.2)	535 (1.8)	2.38 (1.54-3.67)	<0.0001	30 (4.8)	635 (2.0)	2.46 (1.07-5.70)	0.029
Acute hemorrhagic CVA	30 (0.97)	60 (0.2)	4.81 (1.80-12.83)	0.0005	15 (2.4)	75 (0.23)	10.36 (2.96-36.29)	<0.0001
In-hospital mortality	455 (14.7)	1,665 (5.6)	2.88 (2.23-3.73)	<0.0001	150 (23.8)	1,970 (6.2)	4.75 (3.11-7.26)	<0.0001

Values are n (%), unless otherwise indicated.  
 Abbreviations as in Table 2.

patients studied provide valuable data in an understudied population.

## CONCLUSIONS

In this large, propensity-matched cohort, patients with cTCP were at higher risk of complications after PCI, including a 2-fold increased risk of in-hospital mortality. Further studies are needed to mitigate PCI risk in this population.

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## PERSPECTIVES

**WHAT IS KNOWN?** The safety of performing PCI in patients with cTCP is unknown with very limited, and often observational, data.

**WHAT IS NEW?** Our results revealed that patients with cTCP undergoing PCI are more likely to experience procedure related complication, including higher in-hospital mortality.

**WHAT IS NEXT?** A prospective cohort study is needed to confirm these results while studying techniques to mitigate this risk.

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**KEY WORDS** complications, percutaneous coronary intervention, thrombocytopenia

**APPENDIX** For supplemental tables, please see the online version of this paper.