

## Relationship between atherogenic index of plasma and stent thrombosis in patients with acute coronary syndrome

### ABSTRACT

**Objective:** Stent thrombosis (ST) is an uncommon but serious complication in patients undergoing percutaneous coronary intervention (PCI). This study aimed to investigate the effect of atherogenic index of plasma (AIP) on ST.

**Methods:** Among the 10,258 patients who underwent coronary angiography between January 2018 and December 2020, 239 patients who underwent PCI with the diagnosis of acute coronary syndrome (ACS) due to ST were included as the study group (ST group) and 459 patients who underwent percutaneous intervention for ACS and did not have any in-stent lesion as the control group (non-ST group). ST classification was done according to the Academic Research Consortium definition.

**Results:** The mean age of the patients was  $63.3 \pm 10.6$  years (483 male, 69.2%). The groups were similar in terms of characteristic properties, comorbidities, and the drugs being used ( $p > 0.05$  for all). Drug eluting stents were used in 86.5% of the patients. In the ST group, the median time from stent implantation to thrombosis was 285 days. Mean AIP and the ratio of triglyceride to high-density lipoprotein cholesterol (TG/HDL-C) were statistically significantly higher in the ST group than in the controls ( $p < 0.001$  and  $p = 0.018$ , respectively), and a positive correlation was observed between time from stent implantation to thrombosis and AIP and TG/HDL-C ( $r_s = 0.229$ ,  $p = 0.010$  and  $r_s = 0.222$ ,  $p = 0.010$ , respectively). Multivariate logistic regression analysis revealed that stent length, prior ST elevation myocardial infarction, TG/HDL-C, and AIP were independent predictors of ST.

**Conclusion:** AIP is an easy calculable biomarker, and the performance of AIP to predict ST is better than TG/HDL-C.

**Keywords:** acute coronary syndrome, atherogenic index, stent thrombosis

### INTRODUCTION

Stent thrombosis (ST) is a complication that can occur in patients undergoing percutaneous intervention and can seriously affect mortality and morbidity (1, 2). It occurs most often as ST-elevation myocardial infarction (STEMI), and the best treatment method is early reperfusion (3). As a result of the widespread use of drug-eluting stents (DESs), a decrease in the rates of acute and subacute thrombosis and in late and very late thrombosis with new-generation DESs have been noted (4-6). The underlying mechanisms of ST are underexpansion, malaposition, positive remodeling, neoatherosclerosis, stent fracture, and corner dissections (7-10). Calcification of the lesion also increases the risk of complications (11). Insufficient duration of dual anti-aggregant use or the emergence of clopidogrel resistance, long stent use, common atherosclerotic disease, and the high number of stents used are among the mechanisms responsible for ST (12). Increased inflammatory markers such as C-reactive protein and white blood cell count are laboratory parameters that can be detected in patients with ST (13, 14). Because triglyceride (TG)/high density lipoprotein (HDL) cholesterol (HDL-C) ratio (TG/HDL-C) was an independent predictor of cardiovascular events and all-cause mortality in acute coronary syndrome (ACS) after coronary revascularization and the atherogenic index of plasma (AIP), logarithmically transformed ratio of TGs to HDL-C, is superior to TG/HDL-C in studies, we aimed to examine the relationship between AIP and ST on the basis of this hypothesis (15-17).



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### ORIGINAL INVESTIGATION

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## METHODS

This retrospective study included 3,450 patients with ACS who were intervened percutaneously between January 2018 and December 2020. Of these, 2,056 patients with first percutaneous coronary intervention (PCI); 498 patients with prior coronary artery bypass graft; and 95 patients with chronic kidney-liver disease, malignancy, inflammatory disease, obstructive sleep apnea, and missing data in their files were excluded. The study flow diagram is presented in Figure 1. The demographics, medical characteristics, and previous coronary angiography reports of the patients were obtained from their files and health informatics portal, and routine laboratory parameters were examined from the blood samples taken at 6 a.m. (after at least 8 hours of fasting). Stent type (drug eluting or bare-metal), stent diameter and length, drugs used for all patients, time from stent implantation to the occurrence of thrombosis (ST time), and thrombosis type for the ST group were recorded. ST classification was done according to Academic Research Consortium definition (15). The patients were grouped into 3 according to the dose of statins used by them: group 1 (low-dose statin; atorvastatin 10 mg or 20 mg, rosuvastatin 5 mg or 10 mg, or pitavastatin 1 mg), group 2 (atorvastatin 40 mg, rosuvastatin 20 mg, or pitavastatin 2 mg), and group 3 (high-dose statin; atorvastatin 80 mg, rosuvastatin 40 mg, or pitavastatin 4 mg).

Hypertension (HT) was defined as systolic blood pressure (BP)  $\geq 140$  mm Hg, diastolic BP  $\geq 90$  mm Hg, or current use of antihypertensive medications. Diabetes mellitus (DM) was defined as fasting serum glucose  $\geq 126$  mg/dL, hemoglobin A1c  $\geq 6.5\%$ , or the use of blood glucose lowering agents. AIP is a logarithmically transformed ratio of molar concentrations of TGs to HDL-C and is calculated by the formula  $\log \text{TG}/\text{HDL-C}$  ratio.

The study was approved by the Local Ethics Committee of our hospital (13/1/2021, #1230), and the study was performed according to the Declaration of Helsinki.

### Statistical analysis

Kolmogorov–Smirnov test was used to determine whether variables were homogeneously distributed. Continuous variables with normal distribution were expressed as mean  $\pm$  standard deviation and compared using student's *t*-test; continuous variables without normal distribution were compared using Mann–Whitney U test for variables

without normal distribution. Categorical variables were presented as total number and percentages and compared using the chi-square test, and correlations between variables were determined using Spearman correlation test. Binary logistic regression analysis tested variables with  $p \leq 0.05$  in univariate analysis, and the odds ratio (OR) indicates the relative risk of ST. The value  $p < 0.05$  was considered significant, and 95% confidence interval (95% CI) was presented for all ORs. Pairwise comparison of receiver operating characteristic (ROC) curve analysis was performed to determine the ability of AIP and other variables predicting ST. All statistical analyses were performed using the SPSS version 16.0 (SPSS Inc., Chicago, IL, USA) and MedCalc version 19.6.1 (MedCalc Software, Mariakerke, Belgium).

## RESULTS

The demographic characteristics, medical history, and laboratory results of the patients are summarized in Table 1. Whereas 66.2% of the patients in the ST group presented with STEMI and the rest with non-STEMI, the frequency of STEMI in the control group was 57.1% ( $p=0.025$ ). The mean age of the patients was  $63.3 \pm 10.6$  years (483 male, 69.2%). There was no statistically significant difference between the 2 groups in terms of age, DM, HT, sex, and heart failure. Of the patients, 68% were using dual-antiplatelet agents. Although the use of clopidogrel and ticagrelor was different between the 2 groups, the rates of use of prasugrel were similar ( $p=0.040$ ,  $p=0.041$ , and  $p=0.596$ , respectively). Clopidogrel was the predominant antiplatelet agent in patients in the ST group, whereas ticagrelor was replaced by clopidogrel in the control group. In total, 80.2% of the patients had statins and 13.8% had fibrate in their therapy. DESs were implanted in 86.5% of the patients, and 43% of the patients in the ST group had a history of STEMI and 34.5% had a history of non-STEMI-related PCI, whereas these rates were 34.5% and 32.5% in the control group ( $p=0.013$ ). When the laboratory results were compared, it was observed that the mean AIP and TG/HDL-C were statistically significantly higher in the group with ST ( $p < 0.001$  and  $p=0.018$ ). When the groups were compared in terms of statin dose they were using, 8% of patients in the non-ST group were in group 3 and 30% of them were in group 1, whereas the frequency was 20% and 31% in the ST group, respectively, and the difference was not statistically significant ( $p=0.227$ ).

In the ST group, 19% had early (acute or subacute), 40% had late, and 41% had very late ST. The median ST time was 285 days, and we determined a significant positive correlation between ST time and TG/HDL-C and AIP (ST time–AIP:  $r_s=0.229$ ,  $p=0.01$ ; ST time–TG/HDL-C:  $r_s=0.222$ ,  $p=0.01$ ). There was no relationship between HDL-C and ST time and between low-density lipoprotein cholesterol (LDL-C) and ST time (ST time–LDL-C:  $r_s=0.125$ ,  $p=0.149$ ; ST time–HDL-C:  $r_s=0.044$ ,  $p=0.609$ ).

The mean stent diameter was similar ( $p=0.112$ ), but the mean stent length was different between the groups, and the difference was statistically significant ( $p=0.019$ ).

## HIGHLIGHTS

- Stent thrombosis (ST) is a serious complication of percutaneous coronary intervention and generally occurs as ST-elevation myocardial infarction (STEMI).
- Atherogenic index of plasma is a biomarker for atherosclerosis and can be useful for identifying patients at risk for ST.
- The results of this study reveal that stent length, prior STEMI, triglyceride/high-density lipoprotein cholesterol ratio, and atherogenic index of plasma are independent predictors of ST.

Univariate and multivariate logistic regression analyses showed that AIP, TG/HDL-C, prior STEMI, and stent length were the independent predictors of ST (OR=13.403, 95% CI=1.396–128.699, p=0.025; OR=1.305, 95% CI=1.005–1.693, p=0.045; OR=0.392, 95% CI=0.206–0.743, p=0.004; and OR=1.100, 95% CI=1.016–1.191, p=0.019; respectively) (Table 2). The cut-off value of AIP was >0.32 (sensitivity=43.3%, specificity=77.1%, p=0.016), cut-off value of TG/HDL-C was 2.81 (sensitivity=45.1%, specificity=73%, p=0.028), and cut-off value of stent length was 22 mm (sensitivity=75%, specificity=53.7%, p=0.006) for predicting ST. Pairwise comparison of ROC curve analysis revealed that AIP was noninferior

to stent length and prior STEMI, and the ability of AIP to predict ST was better than the ability of TG/HDL-C (Fig. 2, Table 3).

**Table 1. Demographic properties, laboratory results, and angiographic features of the groups**

	ST group (n=239)	Non-ST group (n=459)	P-value
Age, years	62.82±10.64	63.6±10.6	0.365
Sex, male, n (%)	313 (68.3)	170 (71)	0.243
Triglycerides, median (IQR)	162.5 (153)	162 (54)	0.094
LDL, mg/dL	118.21±41.02	118.26±39.12	0.987
HDL, mg/dL	40.61±9.20	41.72±10.26	0.162
AIP	0.31±0.30	0.21±0.17	<0.001
TG/HDL-C	5.66±5.12	4.15±1.93	0.018
CRP, median (IQR)	7.85 (23.95)	4.45 (8.99)	0.853
BNP, median (IQR)	1420 (3557)	1045 (3285)	0.475
Hgb, g/dL	13.58±1.84	13.48±1.85	0.632
WBC, 10 <sup>3</sup> /mL	10.27±3.58	9.67±3.33	0.120
PLT, 10 <sup>3</sup> /mL	252.72±70.88	259.60±88.95	0.477
Creatinine, mg/dL	0.90±0.38	0.83±0.28	0.193
Troponin, median (IQR)	4928.5 (28700)	5462 (15400)	0.713
CK-MB, median (IQR)	14.25 (92.30)	28.60 (89.62)	0.211
Hypertension, %	48	41	0.248
Diabetes mellitus, %	32	27	0.286
Heart failure, %	13.6	14.5	0.836
Beta blockers, %	80	79.4	0.906
ACEI/ARB, %	65	65	0.972
DAPT, %	63.6	70.2	0.228
Clopidogrel, %	77	44.6	0.040
Ticagrelor, %	15.3	46.8	0.041
Prasugrel, %	0	2.1	0.596
Diuretics, %	14.5	14.9	0.922
Statins, Group 1, %	31	30	
Group 2, %	49	62	0.227
Group 3, %	20	8	
Fibrate, %	15	13.3	0.585
Stent diameter, mm	3.09±0.40	2.96±0.37	0.112
Stent length, mm	26.13±6.00	22.97±7.20	0.019

ACEI/ARB - angiotensin converting enzyme inhibitors/angiotensin receptor blockers; AIP - atherogenic index of plasma; BNP - brain natriuretic peptide; CK-MB - creatine kinase-MB; CRP - C-reactive protein; DAPT - dual antiplatelet therapy; HDL-C - high density lipoprotein cholesterol; Hgb - hemoglobin; IQR - interquartile range; LDL, low-density lipoprotein; LDL-C - low-density lipoprotein cholesterol; PLT - platelets count; TG/HDL-C - the ratio of triglyceride to high-density lipoprotein cholesterol; WBC - white blood-cell count

**Table 2. Binary logistic regression analysis**

	Univariate analysis		Multiple analysis	
	OR (95% CIs)	P-value	OR (95% CIs)	P-value
Stent length	1.079 (1.011–1.152)	0.022	1.100 (1.016–1.191)	0.019
AIP	5.382 (2.600–11.138)	<0.001	13.403 (1.396–128.669)	0.025
Clopidogrel	4.127 (1.005–16.947)	0.051	-	
Ticagrelor	0.207 (0.041–1.036)	0.055	-	
Prior STEMI	0.566 (0.458–0.698)	<0.001	0.392 (0.206–0.743)	0.004
TG/HDL-C	1.159 (1.092–1.230)	<0.001	1.305 (1.005–1.693)	0.045
LDL-C	1.000 (0.996–1.004)	0.987	-	

AIP - atherogenic index of plasma; CI - confidence interval; LDL-C - low density lipoprotein cholesterol; OR - odds ratio; TG/HDL-C - the ratio of triglyceride to high density lipoprotein cholesterol; STEMI - ST-elevation myocardial infarction

**Table 3. Pairwise comparison of AIP, TG/HDL-C, prior STEMI, and stent length in ST**

	Difference between		Z statistic	95% CI	P-value
	AUC	SE			
AIP and prior STEMI	0.029	0.0813	0.0361	-0.156–0.162	0.971
AIP and stent length	0.028	0.0772	0.364	-0.123–0.179	0.715
AIP and TG/HDL-C	0.170	0.0762	2.236	0.0211–0.320	0.025
Prior STEMI and stent length	0.025	0.0796	0.316	-0.131–0.181	0.751
Prior STEMI and TG/HDL-C	0.173	0.0874	1.983	0.002–0.345	0.047
Stent length and TG/HDL-C	0.198	0.0775	2.561	0.046–0.350	0.010

AIP - atherogenic index of plasma; AUC - area under curve; CI - confidence interval; SE - standard error; ST - stent thrombosis; STEMI - ST-elevation myocardial infarction; TG/HDL-C - the ratio of triglyceride to high density lipoprotein cholesterol

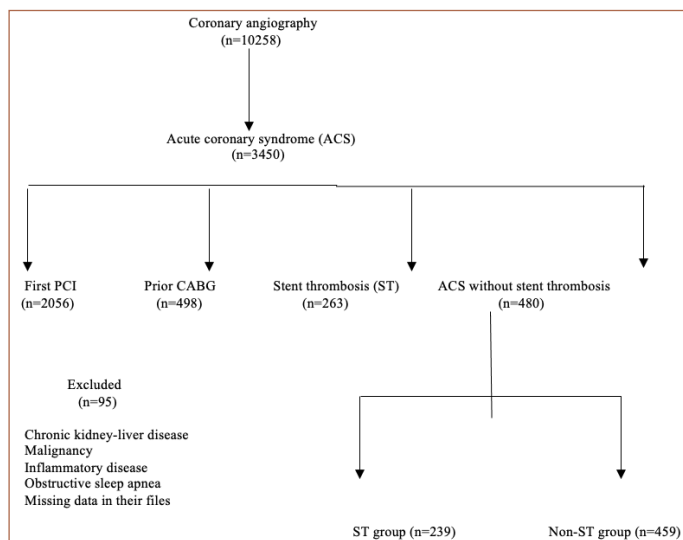


Figure 1. Study flow diagram

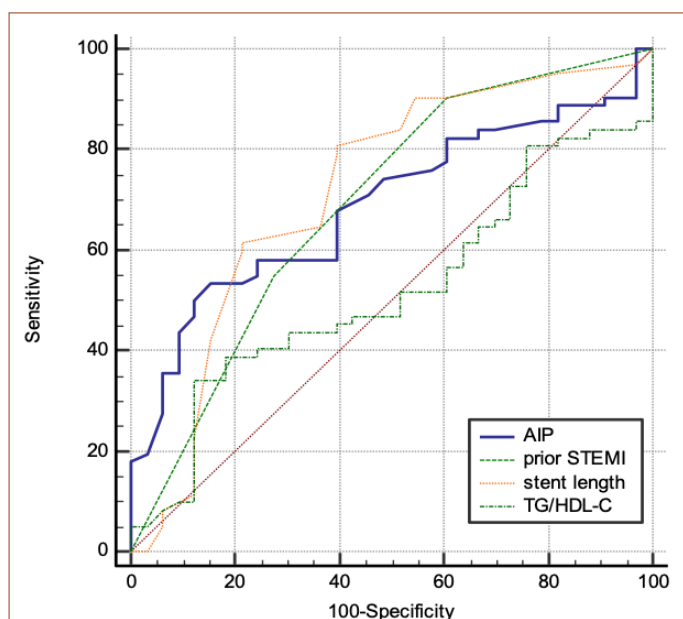


Figure 2. ROC curve analysis of AIP, TG/HDL-C, prior STEMI, and stent length

AIP - atherogenic index of plasma; ROC - receiver operating characteristic; STEMI - ST-elevation myocardial infarction; TG/HDL-C - ratio of triglyceride to high-density lipoprotein cholesterol

**DISCUSSION**

The most important results of this study included the following:

- ST can be detected in approximately 2% of patients who have undergone coronary angiography, and this rate can rise up to 6% in patients with ACS.
- As the use of DESs has increased recently, the frequency of ST is lower.
- LDL-C level and statin use rates are similar in the control and the ST groups. When the patients were grouped according to the statin dose used by them, it was revealed

that a large percentage of the patients received low and medium dose statin treatment.

- Lesion length, AIP, and TG/HDL-C were higher in the ST group than in the control group.
- Logistic regression analyses showed that AIP, prior STEMI, TG/HDL-C, and stent length were independent predictors of ST, and ROC curve analysis revealed that AIP was noninferior to the stent length and prior STEMI and better than TG/HDL-C in predicting ST.

LDL-C, which is known to have an important role in the pathogenesis of coronary artery disease (CAD) and is called bad cholesterol, has also been one of the main drivers of treatment (18-21). The theory that a decrease in serum LDL-C level can reverse this phenomenon is still accepted. Therefore, lowering LDL-C maintains its place as the primary choice in the treatment of CAD. When the pathogenesis is examined in detail, it is seen that LDL-C phenotype B, small dense particles, are more atherogenic. Atherogenic particles begin to appear at the TG level of 90–100 mg/dL and become apparent when TG level reaches 180 mg/dL. AIP is a biomarker that can indirectly provide information about small dense LDL-C and can be calculated using serum TG and HDL levels. It has been found in many studies to indicate the presence and severity of CAD (22). In our study, although the routine LDL-C, TG, and HDL-C values in patients with ST were not different than the controls, AIP and TG/HDL-C levels were higher in the ST group. This result supports that AIP and TG/HDL-C should be among the routine laboratory parameters. Statin doses of the patients included in the study were similar; the fact that most of them use moderate doses may explain the similar LDL-C levels between the groups. It should be kept in mind that this situation may mask the effect of serum LDL-C levels in ST. However, it is known that some of the patients who were given statin treatment in clinical observation did not use the drug and some were treated with low-dose statins because their LDL-C levels were at the limit or that the rate of using statins in high doses owing to side effects is already low. Even that the rate of patients using statins in our study was close to 80% should be considered as a good result.

Apart from the use of statins and the basal lipid levels of the patients, dual antiplatelet treatment (DAPT) and duration are the most important factors to be considered for ST. Early termination of DAPT or development of resistance to any agent, most commonly clopidogrel, leads to a serious increase in the risk of ST (23-25). Contrary to this view, many studies emphasize that long-term use of DAPT does not lead to an increase in the risk of ST (26-28). Of the patients in our study, 68% were taking DAPT, and no difference was observed between the groups in terms of DAPT frequency. In total, 50% of the patients in the ST group had completed the recommended 12-month DAPT period. The rates of using DAPT in early and late ST development groups were 43% and 90%, respectively. Despite the use of DAPT, a significant increase in the incidence of late ST was observed. In our study, the majority of patients in the ST group were using clopidogrel, which may suggest that clopidogrel resistance may have developed.

In addition to factors such as hyperlipidemia, atherogenic dyslipidemia, and DAPT duration, stent type, length, and diameter are among the parameters that can predict ST development (29, 30). In this study, frequencies of late and very late ST were high. AIP is also correlated with the ST time. Supporting the literature, it has been shown in our study that the ST risk increases with the increase in stent length. The small stent diameter, which is reported to be an independent predictor for ST, could not be detected in this study because the diameter of the stents used in the patients included in the study was similar (31, 32).

### Study limitations

This study had a few limitations. First, it was a retrospective, single-center study, and the study population was small. Furthermore, details such as the content of the DES were not included.

### CONCLUSION

In conclusion, this study is important in terms of showing that AIP is equivalent to stent length and prior STEMI and superior to TG/HDL-C in predicting ST in addition to classical ST predictors. At the same time, it suggests that not only lowering LDL-C but also serum TG and HDL levels should be taken into account to prevent ST. If AIP is studied among the routine lipid parameters, it may be necessary to change or expand the treatment options; therefore, this study is valuable in terms of shedding light on multi-center, prospective studies with a high number of participants.

**Conflict of interest:** None declared.

**Peer-review:** Externally peer-reviewed.

**Author contributions:** Concept – Ö.Ö.A.; Design – Ö.Ö.A.; Supervision – Ö.Ö.A.; Fundings – Ö.Ö.A.; Materials – Ö.Ö.A.; Data collection &/or processing – Ö.Ö.A., A.Y., N.Y.K., M.K., S.K.; Analysis &/or interpretation – Ö.Ö.A.; Literature search – Ö.Ö.A., A.Y., N.Y.K., M.K., S.K.; Writing – Ö.Ö.A., M.K.; Critical review – Ö.Ö.A.

### REFERENCES

1. Reejhsinghani R, Lotfi AS. Prevention of stent thrombosis: challenges and solutions. *Vasc Health Risk Manag* 2015; 11: 93-106. [Crossref]
2. Gori T, Polimeni A, Indolfi C, Räber L, Adriaenssens T, Münzel T. Predictors of stent thrombosis and their implications for clinical practice. *Nat Rev Cardiol* 2019; 16: 243-56. [Crossref]
3. Claessen BE, Henriques JP, Jaffer FA, Mehran R, Piek JJ, Dangas GD. Stent thrombosis: a clinical perspective. *JACC Cardiovasc Interv* 2014; 7: 1081-92. [Crossref]
4. Takayama T, Hiro T, Hirayama A. Stent thrombosis and drug-eluting stents. *J Cardiol* 2011; 58: 92-8. [Crossref]
5. de Souza DG, Baum VC, Ballert NM. Late thrombosis of a drug-eluting stent presenting in the perioperative period. *Anesthesiology* 2007; 106: 1057-9. [Crossref]
6. Wang X, Chen X, Sun W, Tian T, Zhou S, Zhang Z, et al. Very Late Stent Thrombosis in Drug-Eluting Stents New Observations and Clinical Implications. *Cardiol Rev* 2019; 27: 279-85. [Crossref]
7. Cuesta J, Rivero F, Bastante T, García-Guimaraes M, Antuña P, Alvarado T, et al. Optical Coherence Tomography Findings in Patients With Stent Thrombosis. *Rev Esp Cardiol (Engl Ed)* 2017; 70: 1050-8. [Crossref]
8. Otsuka F, Nakano M, Ladich E, Kolodgie FD, Virmani R. Pathologic Etiologies of Late and Very Late Stent Thrombosis following First-Generation Drug-Eluting Stent Placement. *Thrombosis* 2012; 2012: 608593. [Crossref]
9. Collet C, Sotomi Y, Cavalcante R, Suwannasom P, Tenekecioglu E, Onuma Y, et al. Coronary stent thrombosis: what have we learned? *J Thorac Dis* 2016; 8: 1398-405. [Crossref]
10. Lee SY, Hong MK. Mechanisms of stent thrombosis: insights from optical coherence tomography. *J Thorac Dis* 2016; 8: E460-2. [Crossref]
11. Kamenik M, Widimsky P. Stent thrombosis in acute coronary syndromes: Patient-related factors and operator-related factors. *Anatol J Cardiol* 2020; 24: 274-9. [Crossref]
12. Sudhir K, Hermiller JB, Ferguson JM, Simonton CA. Risk factors for coronary drug-eluting stent thrombosis: influence of procedural, patient, lesion, and stent related factors and dual antiplatelet therapy. *ISRN Cardiol* 2013; 2013: 748736. [Crossref]
13. Park DW, Yun SC, Lee JY, Kim WJ, Kang SJ, Lee SW, et al. C-reactive protein and the risk of stent thrombosis and cardiovascular events after drug-eluting stent implantation. *Circulation* 2009; 120: 1987-95. [Crossref]
14. Shah B, Baber U, Pocock SJ, Krucoff MW, Ariti C, Gibson CM, et al. White Blood Cell Count and Major Adverse Cardiovascular Events After Percutaneous Coronary Intervention in the Contemporary Era: Insights From the PARIS Study (Patterns of Non-Adherence to Anti-Platelet Regimens in Stented Patients Registry). *Circ Cardiovasc Interv* 2017; 10: e004981. [Crossref]
15. Wan K, Zhao J, Huang H, Zhang Q, Chen X, Zeng Z, et al. The association between triglyceride/high-density lipoprotein cholesterol ratio and all-cause mortality in acute coronary syndrome after coronary revascularization. *PLoS One* 2015; 10: e0123521. [Crossref]
16. Nogay NH. Assessment of the correlation between the atherogenic index of plasma and cardiometabolic risk factors in children and adolescents: might it be superior to the TG/HDL-C ratio? *J Pediatr Endocrinol Metab* 2017; 30: 947-55. [Crossref]
17. Wu TT, Gao Y, Zheng YY, Ma YT, Xie X. Atherogenic index of plasma (AIP): a novel predictive indicator for the coronary artery disease in postmenopausal women. *Lipids Health Dis* 2018; 17: 197. [Crossref]
18. Nelson RH. Hyperlipidemia as a risk factor for cardiovascular disease. *Prim Care* 2013; 40: 195-211. [Crossref]
19. Borén J, Chapman MJ, Krauss RM, Packard CJ, Bentzon JF, Binder CJ, et al. Low-density lipoproteins cause atherosclerotic cardiovascular disease: pathophysiological, genetic, and therapeutic insights: a consensus statement from the European Atherosclerosis Society Consensus Panel. *Eur Heart J* 2020; 41: 2313-30. [Crossref]
20. Paoletti R, Gotto AM Jr, Hajjar DP. Inflammation in atherosclerosis and implications for therapy. *Circulation* 2004; 109 (23 Suppl 1): III20-6. [Crossref]
21. Daniels TF, Killinger KM, Michal JJ, Wright RW Jr, Jiang Z. Lipoproteins, cholesterol homeostasis and cardiac health. *Int J Biol Sci* 2009; 5: 474-88. [Crossref]
22. Guo Q, Zhou S, Feng X, Yang J, Qiao J, Zhao Y, et al. The sensibility of the new blood lipid indicator--atherogenic index of plasma (AIP) in menopausal women with coronary artery disease. *Lipids Health Dis* 2020; 19: 27. [Crossref]
23. Yeh RW, Kereiakes DJ, Steg PG, Windecker S, Rinaldi MJ, Gershlick AH, et al.; DAPT Study Investigators. Benefits and Risks of Extended Duration Dual Antiplatelet Therapy After PCI in Patients With and Without Acute Myocardial Infarction. *J Am Coll Cardiol* 2015; 65: 2211-21. [Crossref]

24. Ielasi A, Al-Lamee R, Colombo A. Stent thrombosis and duration of dual antiplatelet therapy. *Curr Pharm Des* 2010; 16: 4052-63. [\[Crossref\]](#)
25. Ohno Y, Okada S, Kitahara H, Nishi T, Nakayama T, Fujimoto Y, et al. Repetitive stent thrombosis in a patient who had resistance to both clopidogrel and prasugrel. *J Cardiol Cases* 2016; 13: 139-42. [\[Crossref\]](#)
26. Sharma A, Agrawal S, Garg A, Vallakati A, Lavie CJ, Helft G. Duration of dual antiplatelet therapy following drug-eluting stent implantation: A systemic review and meta-analysis of randomized controlled trials with longer follow up. *Catheter Cardiovasc Interv* 2017; 90: 31-7. [\[Crossref\]](#)
27. Pandit A, Giri S, Hakim FA, Fortuin FD. Shorter ( $\leq 6$  months) versus longer ( $\geq 12$  months) duration dual antiplatelet therapy after drug eluting stents: a meta-analysis of randomized clinical trials. *Catheter Cardiovasc Interv* 2015; 85: 34-40. [\[Crossref\]](#)
28. Généreux P, Rutledge DR, Palmerini T, Caixeta A, Kedhi E, Her-miller JB, et al. Stent Thrombosis and Dual Antiplatelet Therapy Interruption With Everolimus-Eluting Stents: Insights From the Xience V Coronary Stent System Trials. *Circ Cardiovasc Interv* 2015; 8: e001362. [\[Crossref\]](#)
29. Suh J, Park DW, Lee JY, Jung IH, Lee SW, Kim YH, et al. The relationship and threshold of stent length with regard to risk of stent thrombosis after drug-eluting stent implantation. *JACC Cardiovasc Interv* 2010; 3: 383-9. [\[Crossref\]](#)
30. Mauri L, O'Malley AJ, Popma JJ, Moses JW, Leon MB, Holmes DR Jr, et al. Comparison of thrombosis and restenosis risk from stent length of sirolimus-eluting stents versus bare metal stents. *Am J Cardiol* 2005; 95: 1140-5. [\[Crossref\]](#)
31. Park KW, Hwang SJ, Kwon DA, Oh BH, Park YB, Chae IH, et al.; Korea Stent Thrombosis Investigators. Characteristics and predictors of drug-eluting stent thrombosis: results from the multi-center 'Korea Stent Thrombosis (KoST)' registry. *Circ J* 2011; 75: 1626-32. [\[Crossref\]](#)
32. Iqbal J, Sumaya W, Tatman V, Parviz Y, Morton AC, Grech ED, et al. Incidence and predictors of stent thrombosis: a single-centre study of 5,833 consecutive patients undergoing coronary artery stenting. *EuroIntervention* 2013; 9: 62-9. [\[Crossref\]](#)