Original Article

The association of the systemic immune-inflammation index and stent thrombosis in myocardial infarction patients after coronary stent implantation—a retrospectively study

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Background: A small number of patients can develop stent thrombosis after coronary stent implantation. Diabetes, malignant tumors, anemia, etc. have been identified as risk factors for stent thrombosis. A previous study confirmed that the systemic immune-inflammatory index is associated with venous thrombosis. However, there are no studies investigating the association between the systemic immune-inflammation index and stent thrombosis after coronary stent implantation, and thus, we designed this study.

Methods: A total of 887 myocardial infarction patients admitted to the Wuhan University Hospital from January 2019 to June 2021 were included. All of the patients received coronary stent implantation and were followed up for 1 year by clinic visit. The patients were divided into a stent thrombosis group (n=27) and a control group (n=860) according to whether or not they suffered stent thrombosis. The clinical features of the two groups were observed, and the receiver operator characteristic (ROC) curve was used to analyze the predictive value of the systemic immune-inflammation index for stent thrombosis in patients with myocardial infarction after coronary artery stenting.

Results: Compared with the control group, the proportion of stent number ≥4 in the stent thrombosis group was significantly higher (62.96% *vs.* 38.72%, P=0.011), and the proportion of patients with a systemic immune-inflammation index ≥636 was markedly increased (55.56% *vs.* 23.26%, P=0.000). The number of stents and the systemic immune-inflammation index were both valuable in predicting stent thrombosis, and the predictive value of the systemic immune-inflammation index was higher, with an area under the curve of 0.736 (95% confidence interval: 0.647–0.824, P=0.000), the best diagnostic value was 636, and the sensitivity and specificity were 0.556 and 0.767. The systemic immune-inflammation index ≥636 and the number of stents ≥4 were independent risk factors for stent thrombosis after coronary stent implantation (P<0.05). Compared with the control group, the incidence of recurrent myocardial infarction was notably increased in the stent thrombosis group (33.33% *vs.* 3.26%, P=0.000), and mortality was significantly higher in the stent thrombosis group (14.81% *vs.* 0.93%, P=0.000).

Conclusions: The systemic immune-inflammation index was associated with the development of stent thrombosis in patients with myocardial infarction after coronary stent implantation.

Keywords: Systemic immune-inflammatory index; coronary stent implantation; myocardial infarction; stent thrombosis

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Introduction

Myocardial infarction is one of the main factors leading to death in middle-aged and elderly people, and coronary stent implantation is one of the primary means of treating myocardial infarction. There were up to 900,000 patients undergoing coronary stent implantation for coronary artery disease in China in 2018. Coronary stent implantation effectively reduces the incidence of recurrent myocardial infarction, but stent thrombosis occurs occasionally (incidence rate of about 2%), mostly occurring within 1 year after surgery (1,2). Stent thrombosis is one of the most dangerous complications after coronary artery stenting, with mortality rates ranging from 5% to 45% (3). At present, smoking, diabetes, malignant tumors, anemia, etc. have been identified as risk factors for stent thrombosis (4-9). The key to preventing stent thrombosis is to identify risk factors and target them accordingly. At present, some scholars used white blood cells and C reactive protein to predict the value of stent thrombosis, but their sensitivity and specificity were limited (10). A previous study confirmed that the systemic immune-inflammatory index is associated with venous thrombosis (11). The stent thrombosis is related to inflammation, and the systemic immune-inflammation index is an indicator of the severity of body inflammation. Therefore, the systemic immune-inflammation index may be related to the development of stent thrombosis. However, there are no studies investigating the association between the systemic immune-inflammation index and stent thrombosis after coronary stent implantation, and thus, we designed this study. We present the following article in accordance with the STARD reporting checklist (available at https://jtd.amegroups.com/article/view/10.21037/jtd-23-363/rc).

Highlight box

Key findings

- The systemic immune-inflammation index was valuable in predicting stent thrombosis in myocardial infarction patients after coronary artery stenting.

What is known and what is new?

- At present, diabetes, malignant tumors, anemia, etc. have been identified as risk factors for stent thrombosis.
- This study confirmed the value of the systemic immune-inflammation index in predicting stent thrombosis in myocardial infarction patients after coronary stent implantation.

What is the implication, and what should change now?

- Further exploration of the molecular mechanism of the systemic immune-inflammation index leading to stent thrombosis is needed.

Methods

General information

A total of 887 myocardial infarction patients admitted to the Wuhan University Hospital from January 2019 to June 2021 were included. All patients received coronary stent implantation and were followed up for 1 year. The patients were divided into a stent thrombosis group (n=27) and a control group (n=860) according to whether or not they had stent thrombosis (all stent thrombosis occurred 1 month after surgery). The inclusion criteria were as follows: (I) acute myocardial infarction; (II) coronary stent implantation; (III) age ≥18 years old; and (IV) complete medical records. The exclusion criteria were as follows: (I) receiving thrombolytic therapy; (II) insufficiency of important organs such as the liver and kidneys; (III) complicated by acute heart failure; (IV) combined with malignant tumors; (V) immune system diseases such as primary immune dysfunction; (VI) chronic infectious diseases; (VII) chronic pain diseases; (VIII) mental abnormalities or cognitive impairment; (IX) failure to cooperate with treatment, transfer, or lost to follow-up; and (X) other major diseases. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by the Ethics Committee of the Wuhan University Hospital (No. 20220042). Individual consent for this retrospective analysis was waived.

Treatment methods

Patients in both groups received coronary stent implantation and were given dual antiplatelet therapy (aspirin enteric-coated tablets, 100 mg/d; Clopidogrel, 75 mg/day) for 1 year postoperatively. Patients with hypertension, diabetes, or hyperlipidemia received antihypertensive, hypoglycemic, or lipid-lowering drugs, respectively. For patients with stent thrombosis, stent catheter thrombolysis was used for treatment, and if necessary, stent thrombectomy was used for treatment.

Observation indicators and follow-up methods

After surgery, monthly follow-up was conducted through

outpatient visits or telephone interviews. The follow-up time was up to 1 year postoperatively. The observation indicators included the following: (I) general information: age, gender, hypertension, diabetes, hyperlipidemia; (II) stent situation: number of coronary artery-implanted stents, stent diameter, and stent length; and (III) biological indicators: D-dimer and the systemic immune-inflammatory index, which was calculated as follows: systemic immune-inflammation index = platelet count × neutrophil count/lymphocyte.

Statistical analysis

Data analysis in this study was performed using SPSS 26.0 (IBM, USA, Chicago), and P<0.05 (two-tailed) was considered to indicate a statistically significant difference. The count data were compared and analyzed by the chi-square test between the two groups and expressed in n (%). The predictive value of the systemic immune-inflammation index for stent thrombosis was analyzed using the receiver operator characteristic (ROC) curve [the optimal diagnostic threshold was the value corresponding to the maximum Yoden index (the point closest to the upper left of the ROC curve)]. Single factor analysis was used to analyze the related factors of stent thrombosis. If P<0.100, the factor would be included in the multivariate regression analysis. Multivariate logistics regression analysis was used to explore the risk factors for stent thrombosis.

Results

Comparison of clinical features of the two groups

Compared with the control group, the proportion of stent number ≥4 was significantly higher in the stent thrombosis group (62.96% *vs.* 38.72%, P=0.011), and the proportion of patients with systemic immune-inflammation index ≥636 was markedly higher in the stent thrombosis group (55.56% *vs.* 23.26%, P=0.000) (*Table 1*).

Predictive value of different indexes for stent thrombosis

The stent number and the systemic immune-inflammation index were valuable in predicting stent thrombosis, among which the systemic immune-inflammation index was the highest, and the area under the curve was 0.736 (95% confidence interval: 0.647–0.824, P=0.000). Age, stent diameter, total stent length, and D-dimer had no significant value in predicting stent thrombosis (P>0.05) (*Table 2* and *Figure 1*).

Risk factors for stent thrombosis after coronary stent implantation

The systemic immune inflammation index ≥636 and the stent number ≥4 were independent risk factors for stent thrombosis after coronary stenting (P<0.05) (*Table 3*).

Effect of stent thrombosis on patient prognosis after coronary artery stent implantation

Compared with the control group, the incidence of recurrent myocardial infarction was significantly increased in the stent thrombosis group (33.33% *vs.* 3.26%, P=0.000), and the mortality was markedly higher in the stent thrombosis group (14.81% *vs.* 0.93%, P=0.000) (*Table 4*).

Discussion

Stent thrombosis is one of the most dangerous complications after coronary artery stenting, with a mortality rate of between 5% and 45%. This study showed that patients with stent thrombosis had significantly increased recurrent myocardial infarction and mortality. In recent years, a large number of scholars have studied the risk factors of various diseases to identify high-risk patients early and intervene promptly (12-15). Therefore, determining the risk factors for stent thrombosis after coronary stent implantation is very important. This present study showed that the systemic immune-inflammation index was valuable in predicting stent thrombosis, and the area under the curve was 0.736 (95% confidence interval: 0.647–0.824, P=0.000). The systemic immune-inflammation index ≥636 was an independent risk factor for intra-stent thrombosis after coronary stent implantation (P<0.05).

The systemic immune-inflammation index comprehensively measures three indicators, namely, neutrophils, lymphocytes, and platelets. An increase in the level of the systemic immune-inflammation index indicates that the level of inflammation in the patient's body is elevated, and it has been confirmed that the systemic immune-inflammation index can better measure the level of inflammation in patients and correlates with disease severity in a variety of diseases (16-20). An increased systemic immune-inflammatory index has been observed

Table 1 The clinical characteristics of the two groups

Category	Stent thrombosis group (n=27)	Control group (n=860)	χ^2 value	P value
Age (years), n (%)			1.639	0.200
≥65	12 (44.44)	281 (32.67)		
<65	15 (55.56)	579 (67.33)		
Gender, n (%)			0.983	0.322
Male	19 (70.37)	524 (60.93)		
Female	8 (29.63)	336 (39.07)		
Hypertensive disease, n (%)	18 (66.67)	610 (70.93)	0.230	0.631
Diabetes, n (%)	12 (44.44)	259 (30.12)	2.533	0.111
Hyperlipidemia, n (%)	24 (88.89)	692 (80.47)	1.194	0.275
Stent number, n (%)			6.440	0.011
≥4	17 (62.96)	333 (38.72)		
<4	10 (37.04)	527 (61.28)		
Stent diameter (mm), n (%)			0.398	0.528
≥3.4	17 (62.96)	489 (56.86)		
<3.4	10 (37.04)	371 (43.14)		
Stent length (cm), n (%)			2.713	0.100
≥15	15 (55.56)	342 (39.77)		
<15	12 (44.44)	518 (60.23)		
D-dimer, n (%)			1.834	0.176
≥0.5 mg/L	26 (96.30)	754 (87.67)		
<0.5 mg/L	1 (3.70)	106 (12.33)		
Systemic immune inflammatory index, n (%)			14.872	0.000
≥636	15 (55.56)	200 (23.26)		
<636	12 (23.26)	660 (76.74)		
Stent type, n (%)			0.001	0.979
Drug coated balloon	15 (55.56)	480 (55.81)		
Drug eluting stent	12 (44.44)	380 (44.19)		
Platelet count, n (%)			1.138	0.286
≥300	16 (59.26)	420 (48.84)		
<300	11 (40.74)	440 (51.16)		
Neutrophil count, n (%)			2.160	0.142
≥70%	17 (62.96)	418 (48.60)		
<70%	10 (37.04)	442 (51.40)		
Lymphocyte, n (%)			0.645	0.422
≥40%	8 (29.63)	320 (37.21)		
<40%	19 (70.37)	540 (62.79)		

Table 2 Predictive value of different indicators for stent thrombosis

Variables	Area under the curve	P value	95% confidence interval	Optimal diagnostic cut-off	Sensitivity	Specificity
Age	0.525	0.660	0.414–0.636	–	–	–
Stent diameter	0.544	0.434	0.451–0.638	–	–	–
Stent number	0.625	0.026	0.551–0.700	3.5	0.630	0.613
Total stent length	0.595	0.092	0.495–0.696	–	–	–
D-dimer	0.561	0.283	0.447–0.674	–	–	–
Systemic immune-inflammatory index	0.736	0.000	0.647–0.824	636	0.556	0.767

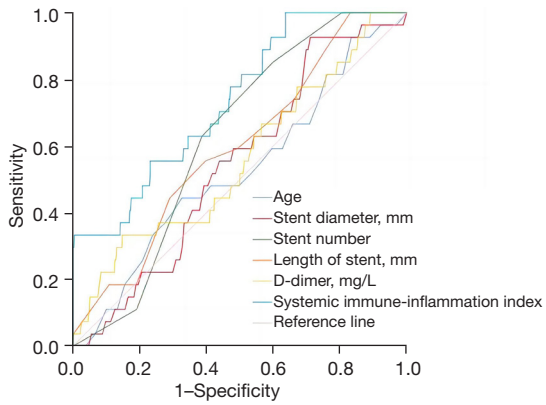

Figure 1 The value of different indicators in predicting stent thrombosis.

in patients with coronary artery disease (21). The systemic immune-inflammation index has also been shown to have a good value for predicting mortality in patients with coronary artery disease (22). An increased systemic immune-inflammation index was also valuable in predicting new-onset atrial fibrillation after coronary artery bypass graft (23). Several other studies have confirmed that the systemic immune-inflammation index correlates with coronary artery disease severity (24-27). The main pathophysiology of myocardial infarction is coronary atherosclerosis, resulting in coronary artery stenosis, which results in blockage and eventually leads to myocardial infarction. Coronary stent implantation is the main means

Table 3 Risk factors for stent thrombosis after coronary artery stenting

Variables	B	S.E.	Wald	P	Relative risk	95% confidence interval
Stent number ≥4	0.908	0.408	4.949	0.026	2.480	1.114–5.520
Systemic immune inflammatory index ≥636	1.359	0.398	11.666	0.001	3.890	1.784–8.483
Constant	–0.087	0.812	0.011	0.915	0.917	–

S.E., standard error.

Table 4 Effect of stent thrombosis on prognosis after coronary stent implantation

Category	Intra-stent thrombosis group (n=27)	Control group (n=860)	χ^2 value	P value
Recurrent myocardial infarction, n (%)	9 (33.33)	28 (3.26)	59.245	0.000
Mortality, n (%)	4 (14.81)	8 (0.93)	37.815	0.000

of treating such myocardial infarction patients; it can restore the blood flow of the myocardium to prevent patients from re-myocardial infarction. These patients often require antiplatelet therapy after surgery; double antiplatelet therapy is a commonly used treatment but stent thrombosis still occurs from time to time. The influencing factors of stent thrombosis include surgical methods, stent types, etc. However, inflammation and the hypercoagulability of blood are also important causes of stent thrombosis. When the systemic immune-inflammation index increases, it signifies that the patient is in an inflammatory state, and the elevated level of inflammation can promote the coagulation process, damage vascular endothelial cells, and promote thrombosis. A previous study also confirmed that an increased systemic immune-inflammation index was a risk factor for acute stent thrombosis after coronary artery stenting (28), which is consistent with our findings. However, this study differs from the previous study in that patients were followed up for 1 year after surgery and explored the effect of the systemic immune-inflammation index on stent thrombosis within 1 year.

Limitations

This was a single-center retrospectively clinical study, which is likely to cause some deviations in the results, therefore the results needs to be further confirmed by multi-center clinical trials. Due to the technical progress in recent years, the incidence of stent thrombosis is relatively low, so the number of patients with stent thrombosis included in this study is relatively small. In addition, this study failed to explore the molecular mechanism through which the systemic immune-inflammatory index leads to stent thrombosis. Moreover, we did not study the predictive value of the systemic immune-inflammation index on the main unconscionable vascular events. Finally, we failed to study the influence of timing of stent implantation on the burden of thrombus during the initial coronary stent implantation.

Conclusions

The systemic immune-inflammation index was valuable in predicting stent thrombosis in patients with myocardial infarction after coronary stent implantation. In patients with an elevated systemic immune-inflammatory index, intervention should be intensified to reduce the incidence of stent thrombosis.

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Footnote

Reporting Checklist: The authors have completed the STARD reporting checklist. Available at https://jtd.amegroups.com/article/view/10.21037/jtd-23-363/rc

Data Sharing Statement: Available at https://jtd.amegroups.com/article/view/10.21037/jtd-23-363/dss

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Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://jtd.amegroups.com/article/view/10.21037/jtd-23-363/coif). The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by the Ethics Committee of the Wuhan University Hospital (No. 20220042). Individual consent for this retrospective analysis was waived.

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