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Original Article

Long-term outcomes of 316 patients with STEMI following coronary stent implantation

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Abstract: Background: Stent thrombosis (ST)-related ST-segment elevation myocardial infarction (STEMI) has very high mortality and poor prognosis. With the extensive construction of the chest pain center in China, the question arises as to whether these special patients will benefit. Methods: From January 2015 to February 2018, 316 patients with STEMI admitted to the coronary care unit (CCU) of Tianjin Chest Hospital after coronary stent implantation were enrolled in this retrospective study. All patients underwent coronary angiography. According to whether STEMI was due to ST, these patients were divided into either a ST group (n=247) or a non-ST group (n=69). The in-hospital mortality and major adverse cardiac events (MACEs), including all-cause mortality, re-ST, target vessel revascularization (TVR), and acute myocardial infarction (AMI) within the 1-year follow-up were compared between the two groups. Results: 78% of cases of STEMI following coronary stent implantation were caused by ST. The in-hospital mortality of the ST group was 0.8% and that of the non-ST group was 1.4% (P>0.05). Forty-two cases had MACEs in the 1-year follow-up, with a higher incidence in the ST group compared to the non-ST group (15.4% vs. 5.8%, P=0.038). The Kaplan-Meier survival analysis showed a lower 1-year event free survival (EFS) in the ST group compared to the non-ST group (84.6% vs. 94.2%, P=0.035). Age over 80-years-old, hypertension, diabetes, hypercholesterolemia, and family history of coronary artery disease (CAD) were all independent risk factors for MACE. Conclusion: ST is the leading cause of STEMI in patients following coronary stent implantation. There was no significant difference in mortality between the ST group and the non-ST group during hospitalization, with a worse prognosis in the ST group during the 1-year follow-up.

Keywords: Stent thrombosis (ST), percutaneous coronary intervention (PCI), major adverse cardiac events (MACEs)

Introduction

As people's living standards constantly improve, with changes in diet structure as well as faster pace of life and work, the incidence of acute myocardial infarction (AMI) has been increasing, with a rising incidence in a young population [1, 2]. The prognosis of AMI has been enhanced over the past decade because of improvements in risk stratification, more widespread use of invasive strategies, advances in antiplatelet agents, and extensive use of secondary prevention strategies such as statins [3, 4]. There are many risk factors for AMI, some of which are controllable while others are not. These include hypertension, smoking, alcohol consumption, hypercholesterolemia, and diabetes mellitus. In European countries, the annual incidence of ST-segment elevation myocardial infarction (STEMI) ranged from 43 to 144 per 100,000, while the reported incidence in the USA was 50 per 100,000 in 2008 [5]. However, STEMI remains one of the leading causes of death worldwide [6], with the in-hospital mortality of unselected STEMI patients varying from 4 to 12% in European countries and reported 1-year mortality among STEMI patients in angiography registries being approximately 10% [5].

The definition of global AMI points out that 4b AMI is related to ST [7]. It was reported that from 2003 to 2010, the incidence of STEMI due to ST increased to 11% from 6% in all patients with STEMI [8], with an annual incidence of 1%-2% [9-11]. It usually presents with cardiac

shock, with an in-hospital mortality rate and a 1-year follow-up mortality rate of 18% and 25%, respectively [12-14].

Primary percutaneous coronary intervention (PCI) is the most important method for the treatment of patients with STEMI. Due to the advantages of relative safety, simplicity, and less pain, it has been widely used in clinical practice [15]. However, compared to other types of coronary heart diseases such as non-ST segment elevation acute coronary syndrome and stable angina pectoris, patients with STEMI have a higher incidence of MACEs after PCI [16]. With the extensive construction of the chest pain center in China, more patients with STEMI received primary PCI [17]. Whether the prognosis of these special patients has been improved, is uncertain and we hope to determine that. Although there have been studies about STEMI caused by ST, they paid more attention to Optical Coherence Tomography (OCT) images but less attention to clinical prognosis [18]. We performed a single-center retrospective study to observe the in-hospital mortality and 1-year prognosis of the patients with STEMI following coronary stent implantation.

Methods

General data

Between January 2015 and February 2018, 316 patients with STEMI who underwent coronary stent implantation and coronary angiography were enrolled, and their data were retrospectively analyzed. The patients included 248 males and 68 females with a mean age of (64.0±10.9) years. Inclusion criteria: (1) Patients with a history of coronary artery stenting and meeting the diagnostic criteria of STEMI [6]. (2) Patients receiving coronary angiography examination during this visit to hospital. (3) Patients in the ST group all met the standard of ST defined by Academic Research Consortium (ARC), including early ST (occurring within 30 days after PCI), late ST (occurring from 30 days to 1 year after PCI), and very late ST (occurring more than 1 year after PCI) according to the time of occurrence [19]. Exclusion criteria: (1) Patients without STEMI, including unstable angina pectoris (UAP) and non-ST-segment elevation myocardial infarction (NSTEMI); (2) Patients with previous coronary artery bypass graft (CABG) surgery; and (3) Patients with a life expectancy less than 2 years. The study was approved by the Ethics Committee of Tianjin Chest Hospital (No. 2018KY-010-01).

Methods

ST-induced STEMI was determined based on imaging characteristics. Patients were assigned to either a ST group or a non-ST group according to the occurrence of ST-induced STEMI and received contemporary standards of care for primary re-PCI including stent re-implantation or balloon angioplasty. All patients were given loading-dose of aspirin 300 mg (then 100 mg/qd), and ticagrelor 180 mg (then 90 mg/bid) or clopidogrel hydrogen sulfate 300 mg (then 75 mg/qd) before re-PCI. Intraoperative intravenous infusion of unfractionated heparin (70-100 U/kg) was performed to maintain the activated clotting time (ACT) of 250-300 s. Then, the decision on stent re-implantation or balloon angioplasty, thrombus aspiration application, tirofiban, drug therapy, CABG, as well as use of intra-aortic-balloon-pump (IABP) was made by two experienced physicians. All patients provided informed consent for the procedure and data collection. Data about patients' baseline clinical features, laboratory, angiography, interventional characteristics, and anti-platelet therapy status were collected. Patients' in-hospital mortality and MACEs, including all-cause mortality, re-ST, TVR, and AMI [20], were recorded by outpatient visits or telephone follow-up 1 month, 3 months, 6 months, and 12 months after the procedure.

Statistical methods

SPSS18.0 statistical software was used for all data analysis. Continuous variables were expressed as means (standard deviation) or median (interquartile range) and compared by Student's t-test or Mann-Whitney U-test as appropriate. Categorical variables were expressed as counts and percentages, and the comparison of these variables was performed with Chi-square or Fisher's exact test. A two-tailed value of $P<0.05$ was considered significant for all comparisons. The EFS of the two groups were estimated using Kaplan-Meier survival curves. Multivariate logistic regression analysis was used to identify risk factors for the occurrence of MACE.

Table 1. Baseline data of patients in both groups

	ST group N=247	Non-ST group N=69	P
Age (year)[a]	64.9±10.3	61.6±11.2	0.070
Male n (%)	190 (76.9)	58 (84.1)	0.202
Cardiovascular risk factors n (%)			
Diabetes	101 (40.9)	24 (34.8)	0.359
Hypertension	150 (60.7)	50 (72.5)	0.074
Hypercholesterolemia	70 (28.3)	27 (39.1)	0.282
Family history of CAD	35 (14.2)	9 (13.0)	0.811
Smoking	104 (42.1)	23 (33.3)	0.354
BMI (kg/m^2)[a]	26.2±2.1	27.0±2.2	0.187
Uric acid, umol/l	333.30±108.22	333.82±86.48	0.972
Homocysteine, umo/l	18.35±9.82	17.30±11.95	0.539
Total cholesterol, mmol/l	4.11±1.03	4.26±1.04	0.334
Triglyceride, mmol/l	1.42±0.77	1.63±1.00	0.132
HDL-C, mmol/l	1.08±0.31	1.03±0.29	0.271
LDL-C, mmol/l	2.70±0.95	2.85±0.97	0.293
VLDL-C, mmol/l	0.32 (0.21-0.47)	0.36 (0.22-0.51)	0.757
Hs-CRP, mg/l	17.96±30.12	14.39±21.61	0.209
BNP, pg/ml	791.5 (350.7-2647.8)	615.8 (235.5-1372.3)	0.090
TNT, ng/ml	2.09 (1.01-7.90)	1.75 (0.36-4.94)	0.117
D-dimer, ug/ml	0.93±0.85	0.86±1.28	0.715
FIB, g/l	3.59±0.88	3.48±0.69	0.206
PLT, *10^9/L	204.97±61.76	219.30±72.60	0.172
Cardiac functional grading (NYHA) n (%)			
<III	207 (83.8)	62 (89.9)	0.212
≥III	40 (16.2)	7 (10.1)	0.212
LVEF n (%)			
LVEF <45%	125 (50.6)	31 (44.9)	0.135
LVEF ≥45%	122 (49.4)	38 (55.1)	0.135

Note: a: Mean ± standard deviation; ST: stent thrombosis; CAD: coronary artery disease; BMI: body mass index; HDL-C: high density lipoprotein cholesterol; LDL-C: low density lipoprotein cholesterol; VLDL-C: very low-density lipoprotein cholesterol; Hs-CRP: hypersensitivity C-reactive protein; BNP: B-type natriuretic peptide; TNT: troponin T; FIB: Fibrinogen; PLT: platelet; NYHA: New York Heart Association; LVEF: left ventricular ejection fraction; P<0.05: There are differences between groups.

Results

General data of two groups

The mean age of 316 patients was (64.2±10.6) years old, with males accounting for 78.5%. Among patients in the ST group (n=247) and the non-ST group (n=69), 78% of STEMI was caused by ST. There was no significant difference between the two groups in age, gender, susceptible factors of coronary heart disease, or clinical diagnosis (**Table 1**).

Re-PCI data of the two groups

In the ST group (n=247), 7 cases received drug therapy, 4 cases received CABG, and 236 cases received re-PCI treatment, with 232 cases of successful treatment, 2 deaths from cardiac shock during hospitalization, and an in-hospital mortality of 0.8%. In the non-ST group (n=69), 7 cases received drug therapy, 3 cases received CABG, and 59 cases received re-PCI treatment, with 55 cases of successful treatment, 1 death from left ventricular free wall rupture during hospitalization, and an in-hospital mortality of 1.4%. There was no difference in the in-hospital mortality between the two groups (0.8% vs. 1.4%, P=0.628). The percentage of balloon angioplasty in the ST group was higher than that in the non-ST group, while the percentage of stent re-implantation was lower (P<0.05) (**Table 2**).

Table 2. Re-PCI data of patients in both groups

	ST group N=247	Non-ST group N=69	P
Emergency intervention (n, %)	210 (85.0)	58 (84.1)	0.844
IABP (n, %)	28 (11.3)	7 (10.1)	0.834
Tirofiban (n, %)	73 (29.6)	17 (24.6)	0.621
Balloon angioplasty (n, %)	85 (34.4)	6 (8.7)	0.000
Stent re-implantation (n, %)	147 (59.5)	53 (76.8)	0.008
D-to B-time (min)[a]	60.2±13.1	59.9±8.8	0.879
CABG (n, %)	4 (1.6)	3 (4.3)	0.138
In-hospital mortality (n, %)	2 (0.8)	1 (1.4)	0.628

Note: a: Mean ± standard deviation; ST: stent thrombosis; IABP: Intra-Aortic-Balloon-Pump; D-to-B: Door-to-balloon; CABG: coronary artery bypass graft; P<0.05: There are differences between groups.

Table 3. 1-year follow-up of patients in the two groups

	ST group N=247	Non-ST group N=69	P
All-cause death (n, %)	7 (2.8)	3 (4.3)	0.525
Re-ST (n, %)	4 (1.6)	1 (1.4)	0.920
TVR (n, %)	13 (5.3)	2 (2.9)	0.414
AMI (n, %)	18 (7.3)	1 (1.4)	0.071
Total MACEs (n, %)	38 (15.4)	4 (5.8)	0.038

Note: ST: stent thrombosis; TVR: target vessel revascularization; AMI: acute myocardial infarction; MACEs: major adverse cardiac events; P<0.05: There are differences between groups.

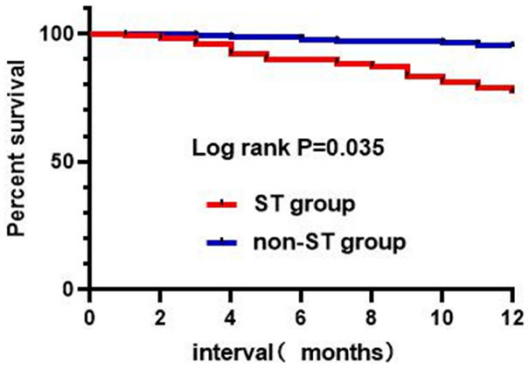

Figure 1. Kaplan-Meier survival curves of the two groups. ST: stent thrombosis.

1-year follow-up of the two groups

A total of 283 cases (89.6%) of 316 patients were successfully followed up. The median of follow-up time was 12 months, and 42 cases of MACE were observed (**Table 3**). The incidence of MACE in the ST group was higher than that of the non-ST group (15.4% vs. 5.8%, P=0.038) (**Table 3**). Kaplan-Meier survival analysis showed that 1-year EFS in the ST group was lower than that of the non-ST group (84.6% vs. 94.2%, P=0.035) (**Figure 1**).

Composition ratio of possible factors between MACE group and non-MACE group

In this study, 42 of the 316 patients developed MACEs during the follow-up period, with an incidence rate of 13.3%. The composition ratios of age over 80-years-old, hypertension, diabetes, hypercholesterolemia, family history of CAD and smoking in the MACE group were significantly higher than those of the non-MACE group (P<0.05) (**Table 4**).

Multivariate logistic regression analysis of MACE occurrence

Multiple logistic regression analysis showed that age over 80-years-old, hypertension, diabetes, hypercholesterolemia, and family history of CAD were all independent risk factors for MACE (P<0.05) (**Table 5**).

Discussion

With the increase in the number of percutaneous coronary intervention (PCI) procedures in China [17], stent thrombosis (ST) has attracted attention. Earlier, it was reported that the in-hospital mortality of STEMI due to ST was far higher than that of denovo lesion-associated STEMI. However, more patients with implanted bare-metal stent (BMS) were enrolled into the study [21], and the proportion of emergency reperfusions was lower. Now with the extensive use of drug-eluting stents (DES), the incidence of ST is increasing [22]. With the extensive construction of the Chinese chest pain center, the proportion of emergency reperfusions has been significantly improved [17]. For the above reasons, we conducted this research, with the purpose of understanding whether the in-hospital mortality and long-term prognosis have been improved in patients with this particular type of STEMI.

The main findings of our study are as follows: (1) 78% of STEMI following coronary stent implantation was caused by ST. (2) There was no significant difference in the in-hospital mortality between the ST group and the non-ST

Table 4. Composition ratio of possible factors of MACE group and non-MACE group

	MACE group N=42	Non-MACE group N=274	P
Age >80 years old [n (%)]	14 (33.3)	3 (1.1)	<0.0001
Male [n (%)]	31 (73.8)	216 (78.8)	0.4632
Cardiovascular risk factors [n (%)]			
Diabetes	33 (78.6)	92 (33.6)	<0.0001
Hypertension	41 (97.6)	159 (58.0)	<0.0001
Hypercholesterolemia	26 (61.9)	71 (25.9)	<0.0001
Family history of CAD	15 (35.7)	29 (10.6)	<0.0001
Smoking	35 (83.3)	92 (33.6)	<0.0001

Note: MACE: major adverse cardiac event; CAD: coronary artery disease.

Table 5. Multivariate logistic regression analysis of MACE occurrence

	β	SE	Wald	P	OR	95% CI
Age >80 years old	5.340	1.142	21.873	0.000	208.524	22.246-1954.600
Diabetes	1.109	0.509	4.753	0.029	3.032	1.119-8.217
Hypertension	2.872	1.367	4.418	0.036	17.678	1.124-257.406
Hypercholesterolemia	1.184	0.565	4.386	0.036	3.266	1.079-9.890
Family history of CAD	1.892	0.509	13.839	0.000	6.631	2.448-17.966
Smoking	0.011	0.673	0.000	0.987	1.011	0.270-3.786

Note: MACE: major adverse cardiac event; CAD: coronary artery disease.

group. (3) Patients in the ST group had more MACEs during the 1-year follow-up.

Because more stents were implanted, the risk of myocardial reinfarction after stenting increases correspondingly. A registered study enrolled 8146 patients implanted with DES and found that the annual incidence of AMI was 0.6% [9]. However, few studies paid attention to the correlation of the special type myocardial infarction with stenting. A Danish registered study enrolled 2300 patients with stent implantation and recorded the occurrence of STEMI during a 4-year follow up, revealing that 62% of STEMI patients after stent implantation were associated with stenting [23]. Our study found that 78% of patients with STEMI after stent implantation were attributed to ST.

Previous studies showed that 70%-80% of patients with ST presented with STEMI, and the in-hospital mortality was significantly higher than that of STEMI caused by denovo lesions [12]. Ergelen et al. found that the in-hospital mortality of AMI caused by ST reached up to 18%, and the mortality of 1-year follow up was 25% [13, 14]. A domestic study enrolling 132 patients with ST found the in-hospital mortality of the ST groups was 6%, and the 3-year mortality was 12.9% [23]. Our study found that the in-hospital mortality of the ST groups was 0.8%, and the 1-year mortality was 2.8% which were lower compared to other studies. The lower mortality may be attributed to the enrolled patients and the difference in reperfusion therapy. The enrolled patients in other studies included possible cases of ST defined by ARC, such as sudden death. All cases enrolled in our study were patients with ST confirmed by coronary angiography, with 84.8% undergoing emergency interventional therapy and an average door-to-balloon (D-to-B) time of 60.1±12.2 min. Strengthening reperfusion therapy and shortening D-to-B time contribute significantly to the decline in mortality.

Is STEMI caused by ST related to a worse prognosis compared to that induced by denovo lesions? There has been little research in the recent ten years. In 2012, a study compared the prognosis of 3305 patients with STEMI resulting from ST to those with denovo lesions [8], and found no significant difference in mortality between the two groups during the 1-month and 2-year follow-ups. Another conclusion was that the recurrence rates of AMI and re-ST were higher in the ST group. However, the control group patients had no stent history, with fewer cardiovascular risk factors in the research [8]. Our study found no significant dif-

ference in the in-hospital mortality between the two groups, but a higher incidence of MACEs in the ST group during the 1-year follow-up. The two groups of patients enrolled in this study had stent history and no obvious difference in cardiovascular risk factors, so the results may be more valuable.

The mechanism of ST after stent implantation is complicated. Early ST may be associated with operative factors, such as poor stent attachment, incomplete lesion covering, poor compliance, and substandard anti-platelet treatment. The factors of late ST included delayed endothelialization, local chronic inflammation, and hypersensitive state [24]. Some studies have shown that local new atherosclerosis may be an important cause for the formation of late ST [25, 26]. In our research, all cases were definite ST patients according the ARC definition, including 9% with early ST, 9% with late ST, and 82% with very late ST.

This study still shows some limitations: (1) The absence of other possible ST as defined by ARC, such as out-hospital sudden death in patients not being recorded, may affect the results of this study. (2) Since it is a single-center and small-sample study, the results need to be further confirmed by multi-center and large-sample studies.

Conclusion

ST is the leading cause of STEMI following coronary stent implantation. There was no significant difference in mortality between the ST group and the non-ST group during hospitalization, but patients in the ST group had a poor prognosis during the 1-year follow-up.

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Disclosure of conflict of interest

None.

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References

[1] Gulati R, Behfar A, Narula J, Kanwar A, Lerman A, Cooper L and Singh M. Acute myocardial infarction in young individuals. Mayo Clin Proc 2020; 95: 136-156.

[2] Zhang Y, Shao T, Yao L, Yue H and Zhang Z. Effects of tirofiban on stent thrombosis, Hs-CRP, IL-6 and sICAM-1 after PCI of acute myocardial infarction. Exp Ther Med 2018; 16: 3383-3388.

[3] Reed GW, Rossi JE and Cannon CP. Acute myocardial infarction. Lancet 2017; 389: 197-210.

[4] Yeh RW, Sidney S, Chandra M, Sorel M, Selby JV and Go AS. Population trends in the incidence and outcomes of acute myocardial infarction. N Engl J Med 2010; 362: 2155-2165.

[5] Ibanez B, James S, Agewall S, Antunes MJ, Bucciarelli-Ducci C, Bueno H, Caforio ALP, Crea F, Goudevenos JA, Halvorsen S, Hindricks G, Kastrati A, Lenzen MJ, Prescott E, Roffi M, Valgimigli M, Varenhorst C, Vranckx P and Widimský P; ESC Scientific Document Group. 2017 ESC guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: the Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC). Eur Heart J 2018; 39: 119-177.

[6] Townsend N, Wilson L, Bhatnagar P, Wickramasinghe K, Rayner M and Nichols M. Cardiovascular disease in Europe: epidemiological update 2016. Eur Heart J 2016; 37: 3232–3245.

[7] Reddy K, Khaliq A and Henning RJ. Recent advances in the diagnosis and treatment of acute myocardial infarction. World J Cardiol 2015; 7: 243-276.

[8] Brodie BR, Hansen C, Garberich RF, Browning JA, Tobbia P, Handran CB, Burke MN, Kadakia H, Stuckey TD and Henry TD. ST-segment elevation myocardial infarction resulting from stent thrombosis: an enlarging subgroup of high-risk patients. J Am Coll Cardiol 2012; 60: 1989-1991.

[9] Daemen J, Wenaweser P, Tsuchida K, Abrecht L, Vaina S, Morger C, Kukreja N, Jüni P, Sianos G, Hellige G, van Domburg RT, Hess OM, Boersma E, Meier B, Windecker S and Serruys PW. Early and late coronary stent thrombosis of sirolimus-eluting and paclitaxel-eluting stents in routine clinical practice: data from a large two-institutional cohort study. Lancet 2007; 369: 667-678.

[10] Stone GW, Moses JW, Ellis SG, Schofer J, Dawkins KD, Morice MC, Colombo A, Schampaert E, Grube E, Kirtane AJ, Cutlip DE, Fahy M, Pocock SJ, Mehran R and Leon MB. Safety and efficacy of sirolimus- and paclitaxel-eluting coronary stents. N Engl J Med 2007; 356: 998-1008.

[11] Mauri L, Hsieh WH, Massaro JM, Ho KK, D'Agostino R and Cutlip DE. Stent thrombosis in randomized clinical trials of drug-eluting stents. N Engl J Med 2007; 356: 1020-1029.
[12] Lemesle G, De Labriolle A, Bonello L, Maluenda G, Torguson R, Xue Z, Satler LF, Lindsay J, Pichard AD and Waksman R. Clinical manifestation and prognosis of early versus late stent thrombosis of drug-eluting stents. J Interv Cardiol 2009; 22: 228-233.
[13] Chechi T, Vecchio S, Vittori G, Giuliani G, Lilli A, Spaziani G, Consoli L, Baldereschi G, Biondi-Zoccai GG, Sheiban I and Margheri M. ST-segment elevation myocardial infarction due to early and late stent thrombosis: a new group of high-risk patients. J Am Coll Cardiol 2008; 51: 2396-2402.
[14] Ergelen M, Gorgulu S, Uyarel H, Norgaz T, Aksu H, Ayhan E, Gunaydın ZY, Isık T and Tezel T. The outcome of primary percutaneous coronary intervention for stent thrombosis causing ST-elevation myocardial infarction. Am Heart J 2010; 159: 672-676.
[15] He H, Chen XR, Chen YQ, Niu TS and Liao YM. Prevalence and predictors of contrast-induced nephropathy (CIN) in patients with ST-segment elevation myocardial infarction (STEMI) undergoing percutaneous coronary intervention (PCI): a meta-analysis. J Interv Cardiol 2019; 2019: 2750173.
[16] Ako J, Morino Y, Okuizumi K, Usami M and Nakamura M. Japanese postmarketing surveillance of clopidogrel in patients with non-ST-segment elevation acute coronary syndrome, stable angina, old myocardial infarction, and ST-segment elevation myocardial infarction after percutaneous coronary intervention in a real-life setting: the final report (J-PLACE Final). Cardiovasc Interv Ther 2016; 31: 101-113.
[17] Hu S, Gao R, Liu L, Zhu M, Wang W, Wang Y, Wu Z, Li H, Gu D and Yang Y. Summary of the 2018 report on cardiovascular diseases in China. Chin Circ J 2019; 34: 209-220.
[18] Adriaenssens T, Joner M, Godschalk TC, Malik N, Alfonso F, Xhepa E, De Cock D, Komukai K, Tada T, Cuesta J, Sirbu V, Feldman LJ, Neumann FJ, Goodall AH, Heestermans T, Buysschaert I, Hlinomaz O, Belmans A, Desmet W, Ten Berg JM, Gershlick AH, Massberg S, Kastrati A, Guagliumi G and Byrne RA; Prevention of Late Stent Thrombosis by an Interdisciplinary Global European Effort (PRESTIGE) Investigators. Optical coherence tomography findings in patients with coronary stent thrombosis: a report of the PRESTIGE consortium (prevention of late stent thrombosis by an interdisciplinary global European effort). Circulation 2017; 136: 1007-1021.
[19] Cutlip DE, Windecker S, Mehran R, Boam A, Cohen DJ, van Es GA, Steg PG, Morel MA, Mauri L, Vranckx P, McFadden E, Lansky A, Hamon M, Krucoff MW and Serruys PW; Academic Research Consortium. Clinical end points in coronary stent trials: a case for standardized definitions. Circulation 2007; 115: 2344-2351.
[20] Sim DS and Jeong MH. Differences in the Korea acute myocardial infarction registry compared with western registries. Korean Circ J 2017; 47: 811-822.
[21] Kubo S, Kadota K, Ichinohe T, Miyake K, Hyodo Y, Otsuru S, Hasegawa D, Shigemoto Y, Habara S, Tada T, Tanaka H, Fuku Y, Katoh H, Fujii S, Goto T and Mitsudo K. Comparison of long-term outcome after percutaneous coronary intervention for stent thrombosis between early, late, and very late stent thrombosis. Circ J 2014; 78: 101-109.
[22] Byrne RA, Joner M and Kastrati A. Stent thrombosis and restenosis: what have we learned and where are we going? The Andreas Grüntzig lecture ESC 2014. Eur Heart J 2015; 36: 3320-3331.
[23] Sarno G, Lagerqvist B, Nilsson J, Frobert O, Hambraeus K, Varenhorst C, Jensen UJ, Tödt T, Götberg M and James SK. Stent thrombosis in new-generation drug-eluting stents in patients with STEMI undergoing primary PCI: a report from SCAAR. J Am Coll Cardiol 2014; 64: 16-24.
[24] Nakazawa G. Stent thrombosis of drug eluting stent: pathological perspective. J Cardiol 2011; 58: 84-91.
[25] Kang SJ, Lee CW, Song H, Ahn JM, Kim WJ, Lee JY, Park DW, Lee SW, Kim YH, Mintz GS, Park SW and Park SJ. OCT analysis in patients with very late stent thrombosis. JACC Cardiovasc Imaging 2013; 6: 695-703.
[26] Otsuka F, Byrne RA, Yahagi K, Mori H, Ladich E, Fowler DR, Kutys R, Xhepa E, Kastrati A, Virmani R and Joner M. Neoatherosclerosis: overview of histopathologic findings and implications for intravascular imaging assessment. Eur Heart J 2015; 36: 2147-2159.