Review

Coronary Stent Thrombosis in 2015: A Comprehensive and Updated Review

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Abstract: Stent thrombosis (ST) is a life-threatening, although quite rare, complication of percutaneous coronary intervention with stenting. According to the Academic Research Consortium, it may be defined as definite, probable, or possible. Moreover, its occurrence after index procedure differentiates between early, late and very late. This complication is associated with high morbidity and mortality and has a very high impact on global health of patients with cardiovascular disorders, therefore a multifactorial approach seems a useful tool for understanding its causes and improving its outcome. In this review we analyze all the most important issues of this disorder beginning from its classification, then we overview the pathophysiological mechanisms and factors implicated and, lastly, we give a glimpse to the future perspective on prevention strategies.

Keywords: stent thrombosis; PCI; long term follow up; DES; BMS.

1. Stent Thrombosis: Case Report

An 84 year-old man with diabetes mellitus was admitted to our interventional cardiology unit for unstable angina. Coronary angiography showed a complex and calcific bifurcation lesion involving left anterior descending (LAD) and diagonal branch (Figure 1A).
After administration of aspirine 100 mg and clopidogrel 600 mg orally, 5000 UI heparin iv., we proceeded to bifurcation revascularization with drug eluting stent (DES) implantation on the main branch (MB)(Figure 1B), renouncing to a second stent implantation on side branch (SB) because it was impossible to recross diagonal branch and patient was asymptomatic.

After 30 minutes, patient experienced new onset of rest angina, and the ECG showed an anterior ST elevation. Emergent coronary angiography showed an acute DES thrombosis with occlusion of LAD, likely due to complex lesion bifurcation pattern and a non adequate expansion of the implanted stent (Figure 1C).

2. Stent Thrombosis: Definition of the Problem

Stent thrombosis (ST) is a serious, although quite rare complication after coronary percutaneous coronary intervention (PCI) and stenting. It usually results in myocardial infarction but may also result in sudden cardiac death [1,2] and is generally caused by an abrupt coronary artery occlusion by thrombus occurring inside or in the proximity of a coronary segment previously treated with a stent. To provide a standardized and consistent definition of ST, the Academic Research Consortium (ARC), a formal collaboration between American and European academic research organizations, was established [3].

According to the ARC criteria, ST is classified in relation to different levels of certainty and to the timing of occurrence after stent implantation. These classifications are summarized in Figure 2 and Table 1. Furthermore, a study [3] based on an autopsy registry of 139 subjects with prior coronary stenting shows that histopathological analysis to detect ST results in high specificity for definite and definite/probable criteria (99% and 83%, respectively), while the sensitivity is lower (18% and 51%, respectively) [4].


<table>
<thead>
<tr>
<th>Definite stent thrombosis</th>
<th>Probable stent thrombosis</th>
<th>Possible stent thrombosis</th>
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</thead>
<tbody>
<tr>
<td><strong>Angiographic confirmation of stent thrombosis:</strong></td>
<td>Clinical definition is considered to have occurred after intracoronary stenting in the following cases</td>
<td>Considered to have occurred with any unexplained death from 30 days after intracoronary stenting until end of trial follow-up</td>
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<tr>
<td>Presence of a thrombus that originates in the stent or in the segment 5 mm proximal or distal to the stent and presence of at least one of the following criteria within a 48-hour time window:</td>
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<tr>
<td>Acute onset of ischemic symptoms at rest</td>
<td>Any unexplained death within the first 30 days</td>
<td></td>
</tr>
<tr>
<td>New ischemic electrocardiographic changes that suggest acute ischemia</td>
<td>Irrespective of the time after the index procedure, any myocardial infarction that is related to documented acute ischemia in the territory of the implanted stent without angiographic confirmation of stent thrombosis and in the absence of any other obvious cause</td>
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<tr>
<td>Typical rise and fall in cardiac biomarkers</td>
<td></td>
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<tr>
<td>Nonocclusive thrombus: Intracoronary thrombus</td>
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<tr>
<td>Occlusive thrombus: TIMI0 or TIMI1 intrastent or proximal to a stent up to the most adjacent proximal side branch or main branch</td>
<td></td>
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<tr>
<td><strong>Definite stent thrombosis: pathological confirmation of stent thrombosis</strong></td>
<td>Evidence of recent thrombus within the stent determined at autopsy or via examination of tissue retrieved following thrombectomy</td>
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</table>

In general clinical practice and with currently available devices, early ST (the one occurring during the first month) occurs variably in about 1% of all PCIs, while after the first month the rate is lower, attesting at 0.2%−0.6% per year (p = 0.003) [5]. Historically, the annual rate of very late ST with first generation DES was of 0.4−0.6% for up to 4 years [6]. If meta-analysis of randomized trials showed similar trends for the risk of early and late ST with DES, as compared with bare-metal stents (BMS) [7,8], several pooled analyses and meta-analyses collectively established an increased risk of very late ST (VLST) with first generation DES (eluting sirolimus or paclitaxel) compared with BMS [9,10,11]. In detail, Ellis et al showed a cumulative rate of ST in 1.28% +/- 0.31% in the DES group and 0.76% +/- 0.23% in the BMS group at 3 years (HR 1.51; p = 0.26). Second-generation DES, such as the zotarolimus- and everolimus-eluting stents, have demonstrated a reduced risk of LST and VLST if compared with the first-generation ones. The results of a recent
registry that compared BMS with first and second-generation DES showed a cumulative incidence of definite ST of 1.5% with BMS, 2.2% with the first-generation DES, and 1.0% with second-generation DES at 3 years [12]. On top of these information, notably 20% of patients who have experienced a ST will have a recurrent episode within 2 years [13].

3. Pathophysiological Mechanisms and Factors Implicated in Stent Thrombosis

ST represents the final pathological result of a multifactorial process, linked to variable and multiple contributing factors (Table 2). Each of these factors could have a different role depending on the stage (early, late, and very late) in which ST develops.

<table>
<thead>
<tr>
<th>Table 2. Risk factors related to ST.</th>
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<tr>
<td><strong>procedural factors</strong></td>
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<tr>
<td>stent malapposition, stent underexpansion, incomplete strut coverage, vulneraple plaques within stents</td>
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<tr>
<td><strong>lesion-related factors</strong></td>
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<tr>
<td>length, location, vessel diameter, complexity</td>
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<tr>
<td><strong>type of stent</strong></td>
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<tr>
<td>drug, polymer, stent-surface</td>
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<td><strong>dual antiplatelet-terapy</strong></td>
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<td>premature interruption, resistance</td>
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<td><strong>comorbidities</strong></td>
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<td>renal failure, diabetes, low ejection fraction, smoking malignancy, trombogenic conditions</td>
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3.1. Procedural factors

Several data show a significant relation between angioplasty-related factors and the risk of early ST. Acknowledged data showed that the most significant predictors of early ST include long stent length, persistent dissection after stenting and low final minimal luminal diameter within the stent [1]. Furthermore, stent underexpansion and malapposition have also been implicated in ST [14] (Figures 3 and 4). On this regard, some authors showed how severe stent underexpansion is a major determinant of acute ST [15]. Finally, multiple, overlapping stents have been associated with higher rates of ST [15,16].
Figure 3. Optical-coherence tomography showing late stent malapposition with thrombus (3 months after implantation).

Figure 4. Optical-coherence tomography showing acute stent malapposition discovered during implantation.
3.2. Lesion–related factors

Lesion site and characteristics play a considerable role for the occurrence of ST. Several studies and large volume registries demonstrated that the coronary artery associated with a higher risk of ST is left anterior descending artery [17,18]. Complex coronary lesions such as chronic total occlusions, bifurcation lesions and lesions in vein grafts are associated with an increased risk of ST [19]. Moreover, bifurcation stenting in the setting of acute myocardial infarction was an independent risk factor for ST [2]. In addition, the clinical indication to PCI (acute vs. stable coronary artery disease, especially in the setting of STEMI) and the extent of coronary disease are directly correlated with the risk of ST, both with BMS or DES [18,20–22].

3.2.1 Factors related to late and very late stent thrombosis

Endothelium plays a pivotal role in the pathogenesis of ST. Exposure of the vessel wall to an offending agent results in intimal thickening with neointimal formation [23]. Instead, if delayed endothelialization of stent struts occurs (with high fibrin exposition), it can represent an important trigger for thrombosis (Figure 5). Data from autopsies of patients died for first-generation DES thrombosis showed a delayed healing of the arterial wall, with persistent fibrin deposition and lower grade of endothelialization. Furthermore, it should be emphasized that the occurrence of ST is not so frequent, although incomplete stent endothelialization is a common finding after first-generation DES implantation. Further, other pathophysiological mechanisms probably contribute to LST or VLST: a local chronic inflammation, or a localized hypersensitivity vasculitis, through lymphocytes, macrophages and eosinophilic infiltrates in tunica intima and media [24]. This reaction, often due to polymer hypersensitivity, could determine a positive remodeling of the vessel wall, resulting in late malapposition and related to a higher risk of VLST [25].

There are also several factors related to the technique of implantation that may increase the risk of late or very late ST. Among them, stent length was a predictor for subacute thrombosis: for each 1mm increase in length, there was a 1.03 times greater risk of thrombosis [26]. Finally, also the adoption of a 2-stent strategy for complex bifurcation lesions, especially with the "crush" technique, was associated with an increased risk of late-occurring ST (4.3% to 9 months) [27] (Figure 6). The evolution of the method with use of the final "kissing balloon", seems to warrant better patient outcomes ensuring a better stent expansion and apposition of the stent struts on vessel wall at the bifurcation [26,28].

3.2.2. In-stent neoatherosclerosis

Despite the reduction in late thrombotic events with newer-generation DES, late stent failure remains a concern following stent placement. In-stent neoatherosclerosis has emerged as an important contributing factor to late vascular complications including very late ST and in-stent restenosis [29,30]. The mechanisms underlying the rapid development of neoatherosclerosis remain
unknown; however, either the absence or an abnormal endothelial functional integrity following stent implantation may contribute to this process [29]. In a post-mortem study, neoatherosclerosis inside the stent occurred significantly earlier in DES lesions and was suggested to be related to VLST [31]. Moreover, the absence of fragments of atherosclerotic plaque does not necessarily mean absence of neoatherosclerosis inside the stents because thrombus aspiration could not fully retrieve the components of the arterial wall [32].

Figure 5. Optical-coherence tomography showing delayed struts re-endothelialization occurring 24 months after implantation of DES.

Figure 6A and 6B. Very late ST occurring 15 months after complex Medina 1,1,1 bifurcation lesion of the left anterior descending artery and first diagonal branch treated with the Crush-stent technique.
3.3. *Factors associated with the type of stent*

Delayed healing is the primary substrate underlying all cases of late DES thrombosis at autopsy series [33]. DES are impregnated with cytotoxic/cytostatic drugs which act locally to inhibit neointimal hyperplasia and subsequently reduce in-stent restenosis. Stents are also coated with polymers that are either biodegradable or durable, to slow down the release of the active drug. Overall, the rates of ST seemed higher with first-generation DES than with BMS while, on multivariate analysis, second-generation DES were associated to similar risk of ST compared with BMS. This finding be partially explained by an improved re-endothelialization with newer DES [12].

The development of a thrombus inside a previously implanted stent can be affected by geometry, material, and coatings. Tada et al through optical coherence tomography (OCT) showed that patients with thin-strut DES were associated with improved rates of stent strut coverage as compared with thick-strut DES at 6–8 months follow-up [12]. Later, biodegradable polymer-coated stents have been developed to enhance biocompatibility and permit a more physiological vascular healing response after stenting. These stents use bio-absorbable polymers that dissolve within a specified time period, with the residual metal scaffolding that gains a similar safety profile to BMS. In the PAINT trial, the tested biodegradable-polymer coated stents releasing either paclitaxel or sirolimus, compared with the same bare-metal platform, showed their effectiveness in reducing combined rates of MACE and the need for re-intervention, showing no increase in ST during the 5-year follow-up. The direct comparison between the DES groups showed that despite superior angiographic end-points for the sirolimus stents, both groups maintained similar event curves throughout the 5-year follow-up [34]. In a meta-analysis biodegradable polymer DES were superior to paclitaxel-eluting stents, but inferior to cobalt-chromium everolimus-eluting stents as regards long-term safety, especially in terms of definite ST [35].

Bio-resorbable everolimus-eluting scaffolds have been proposed to minimize the long-term risks associated with coronary stent implantation, including late and very late ST. In this regard, the four-year clinical results of the first Cohortes of ABSORB studies demonstrate a sustained low rate of MACE (3.4%) without late complications such as ST [36]. However, current research aimed at testing the outcome of these devices in an all-comers population and with long-term follow up will give newer insights on this technology.

3.4. *Dual antiplatelet therapy after stent implantation*

The role of dual antiplatelet-therapy (DAPT) discontinuation is a well recognized factor for the occurrence of early ST and represents its strongest predictor [26,37,38,39,40]. This risk seems to reduce progressively with the time. In 2007 the results of TRITON-TIMI 38, a trial that compared clopidogrel and prasugrel in 13608 ACS patients undergoing PCI, were published [41]. The prasugrel group showed significantly lower rates of myocardial infarction, urgent target-vessel
revascularization, and ST (2.4% versus 1.1%, \( p < 0.001 \)), at the expenses of higher rates of major and life-threatening bleedings (2.4% vs. 1.8%, \( p = 0.03 \)).

The other pivotal trial on newer oral antiplatelets was the PLATO [42] that enrolled 18624 ACS patients and was published in 2009. Here ticagrelor showed an improvement over clopidogrel in the primary endpoint (a composite of vascular death, myocardial infarction, or stroke) (11.7% vs 9.8%, \( p = 0.02 \)). By contrast, no significant difference in the overall rate of major bleeding was showed. Among patients which received a stent during the study, the rate of definite ST was significantly lower in the ticagrelor group than in the clopidogrel group (1.3% versus 1.9%, \( p = 0.009 \)).

Regarding the optimal duration of DAPT after DES implantation, there is still some confusion. Despite the most recent recommendations from various manufacturers on this item seem to suggest to reduce its duration below 1 year, the European guidelines on revascularization recommend that DAPT should be administered for 6 months after new-generation DES in stable coronary-artery disease patients, and for up to 1 year in all ACS patients [43]. On the other hand, the ACC/AHA latest guidelines suggest a period of at least 12 months of DAPT after DES implantation for any clinical indication [44]. The optimal antithrombotic treatment remains a matter of ongoing debate [45].

However, in the Dual Antiplatelet Therapy (DAPT) study [46] 9961 patients were randomized to 12 or 30 months DAPT (clopidogrel or prasugrel). Results showed that the rates of ST and major adverse cardiovascular and cerebrovascular events were reduced significantly among patients with prolonged treatment (4.3% vs 5.9%, \( p < 0.001 \)). By contrast, the rate of moderate or severe bleeding was higher with the prolonged regimen (2.5% vs 1.6%, \( p = 0.001 \)). The 30-month regimen also showed a reduction in total death (0.4% vs 1.4%, \( p < 0.001 \)). The rate of ST was 2% in the 30-month regimen and 1.5% in the 12-month (\( p = 0.05 \)). To note, authors observed an elevated risk of ST and myocardial infarction during the first 3 months after DAPT discontinuation.

### 3.4.1 Patient compliance to DAPT

So much has been written about the role of DAPT for the prevention of ST, however there are certain ancillary issues associated with the use of these medications that are not routinely addressed in the literature, among the others patient compliance to therapy. Predictors of poor adherence to prescribed antithrombotic treatments are comorbidities, low socioeconomic status, inadequate information about the usefulness of therapy at discharge and the lack of adherence to cardiac rehabilitation programs after the procedure. Given that most patients are on a regimen of multiple medications, it is crucial that they recognize the importance of continuing DAPT for the prescribed period. Moreover, dental procedures, surgery and bleeding represent other important causes of premature interruption of antiplatelet therapy without medical advise [47].

### 3.4.2 Resistance to antiplatelet drugs

Antiplatelet therapy resistance is a very serious issue which has gained prominence in the DES era. With regard to biological resistance (failed platelet inhibition), several studies have shown a
large interindividual variability in biological response to antiplatelet therapy, either for aspirin [48,49] or clopidogrel [49,51]. Many authors have suggested an important role of antiplatelet therapy resistance in the incidence of cardiovascular events after PCI. Several reviews have reported a very variable rate of aspirin (5.5–43% of patients) [52,53] and clopidogrel resistance (11%–44%) [51]. However, the real antiplatelet resistance with biological consequences is still a matter of debate due to the many different tests available and because of the lack of consensus among experts.

Patients with low response to antiplatelet therapy, especially clopidogrel, have a higher risk of early cardiovascular events, especially ST [54,55]. It has been shown that 52% of patients with ST had a combined resistance to aspirin and clopidogrel, versus 38% of control patients with stable CAD [56]. Furthermore, some authors have shown that patients with glycoprotein IIb/IIIa receptor polymorphism PLA2 are more frequently resistant to aspirin [50] and this polymorphism is also a predictor for ST (RR 5.26) [57]. Furthermore, there’s a lack of literature data about resistance to newer antiplatelets therapy (prasugrel and ticagrelor): a single case of prasugrel resistance was reported [58].

3.5 Comorbidities

Primary angioplasty could predispose to ST mostly because of possible stent underexpansion, for the following reasons: an inadequate assessment of correct vessel diameter, the common attitude of operators to avoid high inflation pressures in order to reduce the risk of "no-reflow" and finally an high amount of thrombus that will be dissolved within days, thus resulting in inadequate stent apposition to the vessel wall.

There are also other several underlying disease processes associated with an increased incidence of ST, especially chronic renal disease [26,37] and diabetes mellitus [6,59]. Chronic renal failure is frequently associated with microvascular and metabolic alterations predisposing to thrombosis [60]; on the other hand, severe vascular calcifications, often present in these patients, may limit the possibility of adequate stent expansion [61,62]. Diabetic patients have a double risk of ST compared with nondiabetics, especially those requiring insulin [63]. This increased risk may be related both to a pro-thrombotic status, typical of the disease, and to a more extensive and severe amount of coronary atherosclerosis, often associated with suboptimal procedural outcomes [59]. In the Dutch ST Registry the presence of malignancy, left ventricular ejection fraction < 30%, and younger age were associated with an increased risk of ST [16]. Finally, the HORIZONS-AMI trial which enrolled 3,602 STEMI patients undergoing primary PCI either with paclitaxel-eluting or BMS analyzed patients with and without ST in the 2-year follow-up. Patients with ST were younger, had a higher rate of insulin-dependent diabetes, were current smokers, had prior myocardial infarction or PCI, and a higher baseline platelet count [64].
4. Management of Stent Thrombosis

For the operator, it is crucial to firstly recognize the main risk factors for ST, including possible incorrect stent deployment, late-occurring malapposition/aneurysm formation and discontinuation of DAPT. Once ST occurs, its management requires fast vessel reopening with primary PCI.

Currently, a variety of antithrombotic options are available for periprocedural use. The most commonly used agents include unfractionated heparin or low molecular weight heparin ± glycoprotein IIb/IIIa inhibitors. These agents reduce the rates of peri-procedural ischemic and thrombotic events, though these benefits come at the cost of an increase in bleeding complications. Bivalirudin is a direct thrombin inhibitor with a short half-life and linear pharmacokinetics, which results in predictable serum concentrations and anticoagulant effect. Bivalirudin has emerged as an efficacious and safe alternative to heparin plus GP IIb/IIIa inhibitors in both stable coronary artery disease and acute coronary syndrome patients. However, several data show that the use of bivalirudin, although related to significant reduction in major bleeding complications, is associated with an increased risk of definite stent thrombosis [72], even when it’s compared to unfractionated heparin [73]. Furthermore there has been great recent controversy surrounding the role of bivalirudin versus unfractionated heparin in primary PCI despite earlier data based on the results of HORIZONS AMI trial [74]. Recent evidence suggests that a strategy of bivalirudin therapy in primary PCI should be reserved for patients at high bleeding risk [75]. Moreover, in a recent meta-analysis, in patients with STEMI, bivalirudin use resulted in decreased cardiac mortality compared with heparin plus GP IIb/IIIa inhibitors but an increase in definite stent thrombosis at 30 days driven by an increase in acute stent thrombosis [76].

We analyzed a population of 264 patients undergoing primary PCI, comparing three groups with different antithrombotic strategies: 4-hour prolonged bivalirudin infusion vs peri-PCI infusion and heparin plus abciximab. The primary study end point was >70% ST-segment resolution within 90 minutes after PCI. A strategy of prolonged bivalirudin infusion after primary PCI was shown equivalent to a strategy with heparin plus abciximab, with an improvement over standard infusion of bivalirudin in obtaining early microvascular reperfusion [77].

Recently, Lupi et al. have shown in a population of 245 patients treated with primary PCI how an intracoronary bolus of bivalirudin in the infarct related artery improved ST-segment resolution, postprocedural coronary flow and enzymatic infarct size compared with the standard intravenous route [78].

Although data on the outcome after ST is limited, the common high thrombus burden of patients with ST would suggest that aspiration thrombectomy might improve short- and possibly long-term outcomes. Additionally, implantation of a new stent at the time of ST has been associated with adverse long-term outcomes [65,66]. Further studies of ST with long-term follow-up are needed to identify management strategies at the time of ST that will result in improved long-term outcomes.
5. **Outcomes of Stent Thrombosis**

Limited data exist on long-term outcomes of patients with ST. A recent multicentre California registry described the long-term outcomes after angiographically confirmed ST and showed that age, active smoking habitus, diabetes mellitus and bifurcation disease are independently associated with long-term MACE over a five-year follow-up period [67]. Published data highlighted that among patients with acute coronary syndromes who undergo PCI for angiographically documented ST, early ST was associated with the highest in-hospital mortality [65]. Moreover, early, late, and very late ST have significant differences in presentation that may reflect different underlying causes of ST as a function of timing after the initial stent implantation. The mortality risk associated with early ST is high and significantly greater than for patients with late or very late ST [65]. The observed 9.15% in-hospital mortality from STEMI from early ST is higher than the reported 5.5% mortality from the overall population of contemporaneous patients in the NCDR registry presenting with STEMI [68]. Other several trials showed that early ST was more commonly associated with cardiac death at 4 years than later ST (50.8% for early vs 18.5% for late vs 24.0% for very late; p < 0.001) [69,70]. This increased mortality may reflect a predisposition to thrombosis, higher thrombus burden, or complications of the initial PCI [71].

6. **Prevention of Stent Thrombosis and Future Perspectives**

In the last 20 years PCI may be annoverated among the most evoluted operative techniques. The advent of various generations of DES dramatically improved the outcome of this intervention, and afterwards many attempts have been made to reduce the risk of ST. Just as the etiological factors predisposing to ST have been categorized under many subheadings, it may be prudent to do the same when evaluating potentially preventive strategies as well. In this regard we need to act on several modifiable risk factors involved in the occurrence of ST, including proper patient selection, improvement of implantation techniques, development of new antiplatelet drugs and new stent technologies with thinner struts and newer polymers or antiproliferative drugs. When it is possible, patient selection should be based on screening protocols that could help cardiologists to choose the more appropriate stent. In this way outcomes could be improved by reducing the complications due to underlying medical conditions, social and economic factors and nonhaderence to medical therapy. About the proper stent selection, several data, sometimes controversial, have been published. Recent data from the last 2 years have suggested a paradigm shift from the notion that DES are less safe than BMS to the converse. Regarding this, the thin-strut cobalt-chromium everolimus-eluting stents (CoCr-EES) has consistently been associated with the lowest rates of stent thrombosis [79]. Furthermore, the 4-year follow-up results of the ABSORB trial looking at ischemia-driven MACE after implantation of a bioresorbable everolimus-eluting scaffold in patients with de novo coronary artery disease are promising, with no ST reported in these patients [36]. Therefore, a wiser use of
currently available stents and the advent of newer technologies like bioresorbable scaffolds and drug-coated balloons [80], if adopted in selected patients, may drastically reduce the risk of ST.

Regarding procedural issues, the use of intravascular imaging during complex PCI should be considered, in order to make the right choice of stent and to assess its correct expansion, thus reducing the risk of malapposition to the vessel wall. The adoption of an OCT-guided PCI protocol could have a potential for the prevention of ST in some complex cases [81]. The correct duration of DAPT after stent implantation is critical, and should be tailored following the clinical condition of the patient, his anatomical features and the type of intervention.

The optimal duration of dual antiplatelet therapy (DAPT) after drug-eluting stent (DES) implantation is still unclear, and its risks and benefits may vary according to DES generation. Among selected patients undergoing DES implantation, a shorter DAPT strategy seems associated with a reduction in mortality and major bleedings, but at the expense of a higher risk of myocardial infarction and ST. A short duration (6 months) of DAPT appears the safest strategy, while a prolonged duration (24-36 months) reduces thrombotic complications but with an excess in major bleeding complications [82]. Prolonging DAPT requires careful assessment of the trade-off between ischemic and bleeding complications.

However, it is clear that over the type of the device, the antithrombotic therapy or the different risk factors, the patient remains the most unpredictable variable that includes, among social factor, bleeding issues, planned surgeries, baseline conditions, resistance to antiplatelet therapy.

7. Conclusions

ST continues to be an important complication in contemporary PCI-era, often with dramatic consequences: patients presenting with ST still have a poorer prognosis. Although additional mechanisms involved in late and very late ST need to be explored, the amount of information that we have and the technological improvements of the last years have the precise role to limit this event.

Abbreviations

Stent thrombosis (ST); Academic Research Consortium (ARC); Acute coronary syndromes (ACS); Stable coronary artery disease (SCAD); Percutaneous intervention (PCI); Drug-eluting stents (DES); Bare metal stent (BMS); Late ST (LST); Very late ST (VLST); Optical coherence tomography (OCT); Major adverse cardiac events (MACE); Dual antiplatelet therapy (DAPT); ST segment elevation myocardial infarction (STEMI); Left anterior descending artery (LAD); Italian Society of Invasive Cardiology (GISE); Coronary artery disease (CAD); Glycoprotein (GP); cobalt-chromium everolimus-eluting stents (CoCr-EES).

Conflict of Interest

Authors do not have any conflict of interest regarding current manuscript.
References


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