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Innovative therapies for optimizing outcomes of coronary artery disease

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Chapter 7

(Late) Stent Malapposition in the BMS and DES Era

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INTRODUCTION

Since the introduction of drug eluting stents (DES), the incidence of coronary stent restenosis has been significantly reduced. However, the safety of DES became a major issue after several studies associated DES with an increased risk of the rare but often devastating development of late and very late stent thrombosis¹⁻³. Several risk factors have emerged as being important in the development of stent thrombosis, including stent malapposition. Although it is still under debate what the exact role of stent malapposition in this respect is, it appears undeniable that stent malapposition is involved in the development of this severe complication after stent implantation in at least a part of the cases⁴⁻¹⁰.

In this chapter several aspects of stent malapposition will be discussed.

DEFINITION AND CLASSIFICATION

Stent malapposition (SM), also known as incomplete stent apposition, is defined by a separation of at least one stent strut from the intimal surface of the arterial wall with evidence of blood behind the strut, without involvement of side branches¹¹. Stent malapposition may increase the thrombotic risk due to the presence of intraluminal stent struts¹². Although appropriate apposition of the stent to the vessel wall is an important aspect for all stents, it seems to be critical in the case of DESs to ensure antiproliferative drug delivery as well as circumferential vascular support.

Depending on the time point of detection, the following classification can be made; acute if present immediately after the index procedure and late if detected at follow-up.

Furthermore, resolved if present after stent implantation but not at follow up, persistent if present both directly after stent implantation and at follow up and as acquired when the stent is well apposed after the index procedure but SM is detected at follow up. Differentiating between the different forms of SM therefore requires intravascular imaging both at stent implantation and at follow up. Schematically this is shown below as depicted by Hur et al., *Cardiovasc Revasc Med* 2009¹³.(Figure 1)

It is important to realize that the distinction between acute and late SM also implies different pathogenetic mechanisms for these two entities. Furthermore, each form will require a different therapeutic approach, which will be addressed later in the chapter.

PATHOPHYSIOLOGY

The current thought is that acute SM is mostly technique dependent and may result from inadequately sized stent selection or inadequate stent expansion, whereas late acquired SM

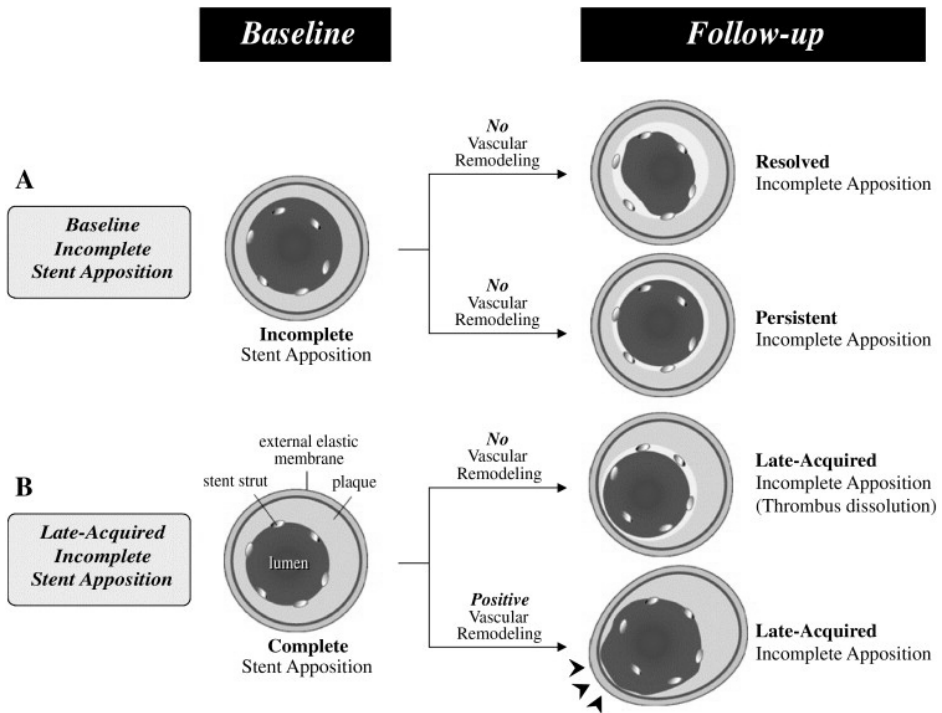


Figure 1. Various types of stent malapposition by Hur et al., *Cardiovasc Revasc Med* 2009.¹³

may result from vessel wall changes in the stented segments that occur during the follow-up period.

Focusing on late SM, several mechanisms have been postulated;¹ positive vascular remodeling of the vessel wall;² decrease in plaque volume;³ chronic stent recoil and⁴ a hypersensitivity reaction to one of the stent constituents.

Positive remodeling is the increase in vascular dimension measured by intravascular imaging using intravascular ultrasound (IVUS) or optical coherence tomography (OCT) by analyzing the change in cross sectional area of the external elastic membrane (EEM) in comparison with the change in plaque volume over time. Several studies reported a greater increase of the EEM compared to the change in plaque volume. Since the size of the stent cannot increase over time, except for the now rarely used self expanding stents, this process will eventually lead to stent malapposition¹³⁻¹⁹. A recent study of Kang et al. used IVUS immediately after intervention and at the 6-month and 2-year of follow-up to evaluate serial vascular changes after DES implantation¹⁹. Their main conclusion was that the development of malapposition was not limited to the first 6 months after implantation as a result of ongoing vascular remodeling even after this period. Therefore, the reported incidence of acquired late SM in previous studies (between 0% and 25%²⁰) maybe underestimated due to relative short-term follow-up periods¹⁹.

Besides an increase of the vascular dimensions, another possible mechanism to stent malapposition is a decrease of the plaque volume. This can be caused by dissolution of a thrombus, present at the stent implantation, mainly seen in patients with acute myocardial infarction^{4, 13, 14, 21, 22}. Furthermore, regression of the plaque under stringent statin treatment can result in creating space between the stent and the vessel wall; however this seems to be only the case in a minority of lesions^{12, 21, 23}.

The pattern of late acquired SM is usually focal and is more often found at the borders of the stents (up to 90%, as reported by Mintz et al.)¹⁷. Additionally, acquired SM is more likely to occur in the relatively disease-free side of the vessel wall, probably because the normal vessel gets more injured during DES implantation and the delayed healing due to the drugs loaded on these stents^{13, 24}. It is therefore more likely that positive remodeling is the most important mechanism for the development of late SM and that the contribution of plaque change is more limited¹³. Although chronic stent recoil could theoretically be one of the causes of SM, it has not been detected using IVUS in patients with SM, making it therefore unlikely to be a factor in the development of SM^{17, 25}.

Another factor of possible influence is the inflammatory response to these stents. In contrast to bare-metal stents (BMS), DES provokes inflammatory responses in animal models, either by a local hypersensitivity reaction to the non-biodegradable polymers of the stent or to the drug released by the stent. Since in a pig model the hypersensitivity reaction only peaks after complete release of the drug, it appears more likely that the polymer is the cause²². Also in human thrombectomy specimens, histopathological signs of inflammation were found, thus supporting the theory that a hypersensitivity reaction underlies the development of SM and stent thrombosis⁵. Furthermore, a growing number of reports documented the formation of coronary artery aneurysm (CAA) after DES implantation. Although the exact mechanism is still unknown, the available evidence suggest that it may be a hypersensitivity-mediated reaction to the delivery polymer²⁶. Studies of related polymers have demonstrated local and systemic hypersensitivity reactions to intravascular polymers^{5, 27-29}. Also, animal studies of DES show that 25% of pigs receiving DES develop eosinophilic infiltrates²⁹. Bare-metal stents have not been demonstrated to elicit such hypersensitivity reactions in human autopsy series of over 400 stents²⁹.

RISK FACTORS

Predictors of acute SM include aneurysmal appearance of the target lesion, larger vessels, lesion calcification, higher patient age, longer lesions and lower balloon pressure^{21, 30}. This is in line with the above statement that acute SM is most likely technique dependent, since these predictors are contributing to the complexity of the target lesion or to the technique itself.

Also for late SM, several factors related to the different proposed mechanisms of SM have been shown to be independent predictors.

The highest incidence of 25% of late SM in DES stented patients has been reported in the MISSION! Intervention study, including patients with acute myocardial infarction²¹. Several studies have shown AMI as an independent predictor of SM^{14,16}. The explanation for this can be found in the second proposed mechanism of SM, since the presence of a thrombus, particularly large thrombus load, which is frequently encountered in AMI may predispose to the occurrence of SM later after the dissolution of the thrombus at the site of stent implantation. Diabetes mellitus has been associated with a lower rate of stent malapposition. The diabetic subpopulation is known to have an increased neointimal growth leading to more restenosis in BMS. Poor glycemic control has been associated with diminished efficacy of sirolimus on smooth muscle cell proliferation, which may explain the lower rate of late SM in these patients^{21,30-32}. Opposing these reports however, another study found a significant greater proportion of diabetic patients in DES cohort with late SM compared to the BMS cohort¹⁴. They reasoned that the proinflammatory role of diabetes may be responsible for a local enhanced inflammatory reaction after DES implantation eventually increasing the risk of late SM.

Another predictor of SM is directional coronary atherectomy (DCA) before stenting. The higher incidence of late SM in DCA before stenting might be explained by the fact that aggressive debulking with DCA is associated with deep vessel injury and promotes more positive remodeling¹⁵.

Despite angiographic optimization with high pressures and adequately sized balloons, malapposed stent struts are frequently found in complex coronary lesions and more often following the implantation of Cypher Select stents which have a thicker stent strut and closed cell design³³. Other factors associated with the lesion complexity, such as longer stent length, C-type lesions and overlapping stents, larger vessel reference diameter, have also been associated with an increased risk of SM^{14,16,21}. Also, chronic total occlusion (CTO), defined by the absence of antegrade flow or only minimal flow of contrast distal to the occlusion during coronary angiography before stent implantation, has been reported as an independent predictor of late SM after DES implantation¹⁶.

Patient age has been associated with the risk of SM, although not consistently. In a recent report of Steinberg et al., subjects with acute SM are older than those without acute SM, whereas (younger) age was the only independent predictor of late acquired SM. Their explanation for this last finding is that most of the other reported risk factors for SM were excluded from their study³⁰.

As will be discussed in the next section, also the stent type is associated with the risk of late stent malapposition. All risk factors are summarized in Table 1.

Table 1. Risk factors for stent malapposition

Clinical factors	Procedure related factors
Acute myocardial infarction (L)	Drug eluting stent (L)
Absence of diabetes mellitus (L)	Lower maximum balloon pressure (A, L)
Chronic total occlusion (L)	Larger vessel reference diameter (A, L)
Patient age (A,L)	Longer stent length (A, L)
	Overlapping stents (A, L)
	C-type lesion (A,L)
	Directional coronary arterectomy (L)

(A) Risk factor for acute stent malapposition, (L) Risk factor for late stent malapposition

BMS VERSUS DES

Acute SM is frequently observed both after DES and BMS implantation^{21,30}. Considering the mechanism of acute SM, a similar incidence in both DES and BMS is in the line of expectation. Late SM is rare after BMS and seems to be related to stent under-expansion in most patients²¹. Although not all, several studies have reported that late SM is much more common after DES implantation^{6,14,21,24,30,34}. A recent meta-analysis of Hassan et al, aimed on clarifying this, included 2453 patient after BMS implantation and 2195 patients receiving DES from a total of 17 studies²⁰. The incidence of late acquired SM after DES varied between 0% and 25%, whereas after BMS implantation the highest reported incidence of SM was 6% at 6 months. They concluded that the risk of late acquired SM was 4.4-fold higher in patients after DES implantation compared to those with BMS. A comparison of paclitaxel- with –limus-eluting stents did not yield significant findings. The higher risk of SM after DES is likely due to the effects of the drugs on the vessel wall, resulting in positive remodeling^{20,21} In BMS the lumen changes at the site of SM are more related to plaque burden²¹.

Furthermore, Hassan et al. also conducted a meta-analysis on the risk of (very) late stent thrombosis in patients with late SM. They concluded that the risk of stent thrombosis is 6.5 times higher (95% confidence interval 1.34-34.91) in patients with late SM compared with those without late SM²⁰.

So late SM appears to be more frequent after DES implantation and is associated with an increased risk of late stent thrombosis. After adequately lowering the incidence of restenosis, the primary complication of balloon dilatation and BMS implantation, we may have created a potentially more devastating obstacle with late SM and subsequent stent thrombosis in DES.

DIAGNOSTICS

Although intravascular ultrasound (IVUS) has been usually regarded as the gold standard for *in vivo* assessment of stent strut apposition to the vessel wall, more accurate evaluation of stent strut apposition cannot be successfully performed in some lesions with minimal stent malapposition due to the limited resolution capacity in IVUS (100-150µm)³⁵. Furthermore, this limited axial resolution of IVUS makes it virtually impossible to discriminate between atherosclerotic plaque and thrombus²¹. It also has problems with stent-related artifacts¹², possibly resulting in underestimation of the evaluation of re-endothelialization. SM is associated with a decreased re-endothelialization after DES implantation²², which is in turn associated with an increased risk of stent thrombosis⁷.

Optical coherence tomography (OCT) is a relatively new imaging modality. In contrast with IVUS, OCT visualizes intra-coronary features using near-infrared light instead of ultrasound, leading to a far better axial resolution, able to resolve detail up to 10 µm¹². Multiple studies investigating stent malapposition using OCT and comparing it with IVUS findings have already been published. There is evidence that minimal stent malapposition which is not detectable by IVUS may disappear or decrease in follow-up OCT evaluation³⁵ and SM without complete re-endothelialization is associated with presence of OCT-detected thrombus¹². More detailed information provided by OCT imaging also led to the conclusion that rate of stent strut coverage and malapposition were significantly different among different DES types and among the type of clinical presentation. More SM was found in sirolimus-eluting stents and paclitaxel-eluting stents compared to zotarolimus-eluting stents and the rate of SM was higher in patients presenting with acute coronary syndrome compared to those with stable angina pectoris^{36,37}. Despite the clear advantages of OCT over IVUS, some disadvantages should also be mentioned. The cost of the device is higher than that of IVUS and the device is not available in every center. A major limitation of OCT is the requirement of a blood-free imaging field because red blood cells scatter light. This can be achieved with a continuous saline flush administration or with a temporary balloon occlusion catheter, leading to transient ischaemia which is not desirable and or tolerated in all cases. Another disadvantage of OCT is poor penetration into non-transparent tissues thus allowing evaluation of only superficial structures including coronary plaques with thin-caps³⁸.

TREATMENT

The most important and potential devastating complication of SM is stent thrombosis. The mechanism by which SM may contribute to stent thrombosis remains unclear. SM may serve as a local nidus for thrombus formation, allowing fibrin and platelet deposition. Delayed re-endothelialization, impaired vasomotion and also involvement of chronic inflammation

and delayed healing, leading to tissue necrosis around the stent, all contribute to creating a thrombogenic environment^{20,23}.

When performing intravascular imaging directly post stent implantation, immediate action can be taken after diagnosing acute stent malapposition by re-dilatation of the target lesion, further expanding the stent. Considering dissolution of a jailed thrombus as a possible mechanism of SM, it may be important to effectively remove thrombotic material prior to stent implantation. Unfortunately, it has been shown that immediate post-intervention IVUS showing no malapposition does not guarantee an uneventful course after DES implantation⁸. After diagnosing stent malapposition during follow-up, defining a proper treatment strategy is difficult, especially when the patient is asymptomatic. The main goal should be off course to prevent stent thrombosis. One option could be pharmacological with long-term double anti-thrombotic treatment¹¹. However, this will also lead to a continuous increased bleeding risk and it is evident that SM may also persist for years without leading to complications, creating a risk of exposing patients to unnecessary side effects of the treatment without having the benefit of it. Another option could be dilatation and implantation of a second, larger, stent³⁹.

FUTURE PERSPECTIVES

It goes without saying that all the, until now, unanswered questions about the mechanism of stent malapposition, its association with stent thrombosis and the best treatment of SM are to be clarified in future larger studies.

One of the current developments in this field is the development of a bioabsorbable stent. The theoretical advantages of such a stent is that it might have less potential for late stent thrombosis because there will eventually be no foreign material exposed to the bloodstream. Also problems with later surgical revascularization, eliminating vasomotor reactions and interference with imaging techniques could be prevented⁴⁰. However, this remains to be proven and also partial/unequal stent dissolution may perhaps cause problems.

Lately, there have been some ongoing clinical trials addressing the use of self-expandable nitinol stents in the treatment of AMI patients with long-term follow-up IVUS and OCT⁴¹⁻⁴³. The hypothesis generated in these trials was that the self-expandable stents would better accommodate to early changes in the vessel wall (thrombus dissolution and vasodilatation) with better apposition due to its self-expandable properties. Initial results have shown that these stents provided 20% increase in lumen area with perfect apposition on 3 days follow up OCT. However, long-term (safety) results are still lacking.

CONCLUSION

Stent malapposition appears to be a relative common finding after stent implantation, especially diagnosed using OCT which has a superior resolution compared to IVUS. The incidence of late SM is higher after DES implantation, mainly caused by positive vascular remodeling. Although SM is associated with stent thrombosis, this is a relatively rare complication. Since the incidence of SM is vastly greater than that of stent thrombosis, SM is not necessarily *sine qua non*, but more likely only one factor in a complex system. Nevertheless, stent thrombosis is a devastating complication and must be prevented where possible. Therefore, further research unraveling the exact mechanisms of stent malapposition and stent thrombosis and possible treatments will continue in future clinical trials.

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