Original article

Influence of parent vessel – side branch distal angle on restenosis rates after main vessel stenting in coronary bifurcation lesion

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Abstract

Background and aim: This study explores predictors of side branch (SB) compromise in the main vessel only stenting group and its influence on long-term follow-up of the patients.

Methods: We hypothesised that the geometric factors determining plaque distribution in branching regions influence SB compromise. Angiographic analysis of bifurcation lesions (all Medina types) was performed before, immediately after, and 9-12 months after the procedure. Control angiography was performed when clinically indicated. Specific attention was given to the influence of angle alpha – the angle between main vessel and SB axes.

Results: Fifty-five patients (62 lesions) formed the study group. The LAD lesions were dominant (73%). Drug-eluting stents were used in 48% and kissing balloon inflation in 31%. The value of angle alpha was associated with significant SB stenosis. There was significant worsening of ostial SB stenosis (from 48% to 69%) after main vessel stenting, with the only independent predictor angle alpha. For SB ostial MLD independent predictors were angle alpha, SB vessel diameter and MB reference diameter. Predictors of SB occlusion (6.5%) were angle alpha <30° and age >82 years. At follow-up (mean 11 months) SB restenosis rate was 52%, but was associated with symptoms only if the main vessel was affected (8/55, 15%). Angle alpha and main vessel reference diameter, main branch minimal diameter after stenting and stent type were predictors of target vessel revascularisation rate (25%).

Conclusion: Angle alpha predicts SB compromise after main vessel stenting and is the main predictor of restenosis in the main vessel.

Key words: coronary stents, bifurcation lesions, carina displacement

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Background

The geometry of a branching point is complex and responsible for atherosclerotic plaque development [1-12]. It is logical that factors determining native plaque development will come into play also in the vessel reconfiguration resulting from stent implantation. These changes are independent from any plaque changes [13-16]. If an inherently curved pipe is forcefully straightened that will significantly deform its walls.

At a mean time side branch (SB) compromise [some surrogate of SB closure and significant ostial stenosis after main vessel (MV) stenting] was considered as an unpredictable event [17-19]. Among the numerous factors cited in different studies probably the most frequently mentioned are origin of SB from MV lesion and presence of significant ostial narrowing [20, 21]. However, as has been shown by Koo et al. [22] 'angiographically' significant ostial branch stenosis most frequently does not mean 'functionally' significant stenosis. At follow-up, despite higher frequency of SB restenosis this does not influence patient outcomes [17, 18, 22-28]. The restenosis in the MV, on the other hand, influences patient outcome, because of the larger myocardial territory at risk.

We hypothesised that factors acting on plaque formation and determining vessel geometry would influence acute and long-term procedural outcome. The aim of the present study was to identify predictors of SB compromise as a result of only MV stenting and its influence on bifurcation restenosis as a whole.

We assumed that the main mechanism of SB stenosis is displacement of the carina of bifurcation, which acts as a gate closed from main branch (MB) stent struts [13]. Using

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a simplified two-dimensional presentation describing reconfiguration of branching points from stent implantation, we found that the resulting percentage of ostial diameter stenosis is equal to the cosine of the angle alpha [13]. This angle permits one to predict percentage diameter stenosis and minimal lumen diameter at the SB ostium after MV stenting. It is a factor that governs carina position and parent vessel flow separation between branches. As such, it determines hydrodynamic conditions at the flow divider region. The full theoretical description can be found elsewhere [13-16].

Methods

Angiographic analysis

Quantitative angiographic analyses were performed using commercially available software (Medis QCA version 5.0 and Dicom Works version 3.1.5b) for angulation assessments. Catheter calibration was used in all cases.

Bifurcation lesions were classified according to Medina using an index of 1 for stenosis greater than 50% and an index of 0 for no stenosis [29]. Analyses were performed for SB diameters greater than 2 mm. The MV before the SB, the MB, and the SB were analysed separately. Reference vessel diameter and minimal lumen diameter before and after stenting, acute lumen gain (minimal lumen diameter after stenting minus minimal lumen diameter before stenting), acute lumen gain at the proximal and distal limb of bifurcation (vessel diameter at proximal or distal limb after stent implantation minus minimal lumen diameter in MV or MB), and the percent diameter stenosis in the MV, MB, and SB before and after stent implantation were calculated.

Measurement of angle alpha was performed as follows (Figure 1). First, the projection with the widest opening between branches was identified without any overlap between vessels. When in doubt of the double contour, the projection was discarded. A line was traced parallel to the MV axis and through the apex of the flow divider (apex of carina) (Figure 1, white lines), and a line parallel to the internal contour of the SB was traced and crossed with the first line: the resulting angle is alpha. The angle alpha was also determined before and after stent implantation.

Procedure protocol

A single stent implantation in MV across the SB is a default strategy for the institution. Heparin was given in a standard dose (100 U/kg, i.v.) at the start of the procedure. If the patient was not pre-treated with clopidogrel, a 600 mg dose of clopidogrel was also administered orally at the start of the procedure. Application of GP 2B/3A inhibitor was left at the discretion of the operator.

Predilatation of one or both branches was performed if initial critical stenosis existed or there was visible calcium. Final kissing balloon inflation or sequential balloon inflation was strongly suggested if more than 85% ostial SB stenosis occurred after MV stenting or flow diminished. In most cases, direct stenting was the preferred strategy. Use of drug-eluting stents in bifurcation lesions was recommended for all diabetic patients and for patients with a vessel diameter below 3.0 mm. Measurement of myocardial necrosis markers (CK-MB and/or troponin I) was performed the day following the procedure.

Follow-up

All patients were followed up to 9-12 months. In case of typical symptoms (effort angina with typical characteristics) and/or dynamic changes in ECG associated

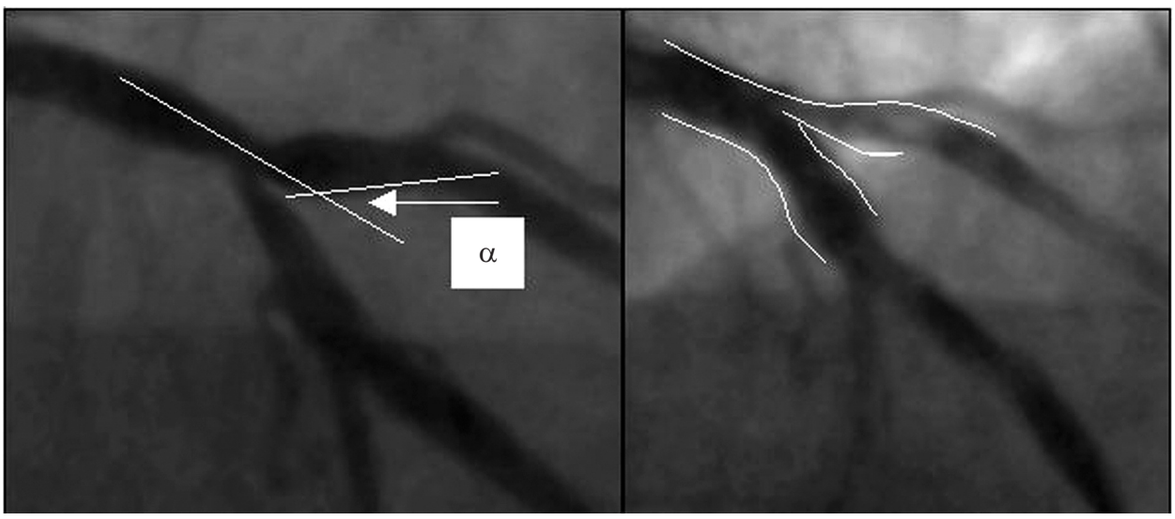

Figure 1. Angle alpha measurement corresponds to the angle between the main vessel axis before the SB, and the internal side of the SB wall, extending up to the flow divider. In this patient, angle alpha is 41°, with a predicted 75% diameter stenosis after stenting. Actually observed is 62%, because of visible stent underexpansion at the carina region

with symptoms repeat coronary angiography was performed. If there were some non-typical symptoms or some doubt about occurrence of angina, dobutamine stress echocardiography or exercise test was performed. Finally, if the test was positive or doubtful, the patient underwent repeat coronary angiography. Several parameters were recorded during the follow-up angiography: lesion restenosis – in MB diameter stenosis more than 50% or more than 70% diameter stenosis in SB, analysed separately; target vessel restenosis – occurrence of >50% diameter stenosis, more than 5 mm proximally or distally from previously implanted stent; target lesion revascularisation (TLR) – intervention in zone (stent region ±5 mm of stent endings) of previously implanted stent or repeat stent implantation; target vessel revascularisation (TVR) – angioplasty in same vessel, but in different location from initial; stent thrombosis – defined as angiographically detected thrombus according to Academic Research Council (ARC) definition.

Statistical analysis

All data are presented as means ± one standard deviation. Differences between groups were examined with paired or unpaired t-test as appropriate. Analysis of Variance (ANOVA) was used for multiple comparisons of data. Multiple regression analysis was used to identify predictors of SB ostial stenosis and closure. A χ^2 test was used to compare qualitative data. Significance was determined by p values less than 0.05. Receiver operator characteristic curve analysis was performed to identify cut-off points for predictor parameters. All calculations were performed using SPSS 13.0 for Windows (SPSS Inc., Illinois).

Table I. Characteristics of patient cohort

Parameter	N (%)
Age [years] (mean ± SD)	66±11
Gender – males	36 (65)
Stable angina	38 (69)
NSTEMI or unstable angina	9 (16)
STEMI	6 (11)
Hypertension	39 (71)
Elevated cholesterol or statin treatment	42 (76)
Diabetes	18 (33)
Smoking	32 (58)
Family history	22 (40)
Previous myocardial infarction	17 (31)
Previous PCI	28 (51)
Previous CABG	7 (13)
Obesity	31 (56)

Abbreviations: NSTEMI – non-ST elevated myocardial infarction, STEMI – ST elevated myocardial infarction, PCI – percutaneous coronary intervention, CABG – coronary artery bypass grafting

Results

Demographics and acute post-procedure results

During a 5-month period (from December 2006 – April 2007) 126 consecutive patients with treated bifurcation lesions were extracted retrospectively from the database. Seventeen (13%) were treated with two stent techniques. In 16 (13%) patients, records were with unacceptable quality for analysis. In 38 (30%) patients, there were no optimal projections for opening of bifurcation angles. Finally, 55 (44%) patients formed the study population.

In all patients, only stenting of the MV was performed. Demographic and procedural characteristics of the patient population are presented in Tables I and II. Approximately half of the patients had triple vessel disease (47%) and presence of single vessel disease was small (10%). Similarly to previous studies, left anterior descending artery lesions were dominant.

Table II. Procedural characteristics

Affected vessel – lesions	N (%)
LAD / Diagonal	43 (73)
LCX / OM	2 (3)
RCA – PD / PL	6 (10)
LM – LAD	5 (8)
LM – LCX	7 (11)
Main vessel predilatation	25 (40)
Balloon size diameter	2.57±0.43
Balloon length	17±5
Inflation pressure [max., atmospheres]	12±4
Side branch predilatation	19 (31)
Balloon size diameter [mm]	2.32±0.30
Balloon length [mm]	15±4
Inflation pressure	10±2
Stent used	60
DES	29 (48)
Co-Cr	11 (18)
SS	20 (33)
Stent diameter [mm]	3.39±0.35
Stent length [mm]	22±9
Implantation pressure [max., atmospheres]	15±2
Kissing balloon inflation	19 (31)
Sequential balloon inflation – both branches	1 (2)
Main vessel	5 (8)
Side branch	8 (13)
IIB/IIIA inhibitor	9 (16)

Abbreviations: LAD – left descending artery, LCX – left circumflex artery, OM – obtuse marginal branch, RCA – right coronary artery, PD – postero-descending branch, PL – postero-lateral branch, LM – left main stem, DES – drug-eluting stent, Co-Cr – cobalt chromium alloy stent, SS – stainless steel stent. Pressure is expressed in atmospheres and other parameters are in millimetres

Post-procedural angiographic characteristics are presented in Table III. There was a significant worsening of ostial SB % diameter stenosis (from 48% to 69%, p <0.001) after stent implantation. Using multiple linear regression analysis with backward stepwise elimination procedure, the only independent predictor of % diameter stenosis at SB was angle alpha (R=0.902, p <0.001, b=–0.011, p <0.001). By the same model for determination of predictors of SB ostial minimal lumen diameter, the only independent predictors were angle alpha (b=0.031, p <0.001), SB vessel diameter (b=0.608, p <0.001) and MB reference diameter (b=–1.006, p=0.04). There were four occluded side branches after the baseline procedure (6.5%). There were no independent predictors for branch occlusion in logistic regression analysis. In multiple ROC curve analysis only angle alpha below 30 degrees (meaning predicted % diameter stenosis = 87%, sensitivity 75%, specificity 91%, p=0.003) and age more than 82 years (sensitivity 100%, specificity 87%, p=0.002) were associated with occlusion.

We hypothesised that angulations play a significant role in plaque development in bifurcation lesions. Indeed, there was a significant difference between pre-intervention angle alpha values in groups with or without significant SB ostial stenosis before stenting (Figure 2).

Table III. Angiographic characteristics. All lengths and diameters are in millimetres

Angiographic characteristics	Before stent	After stent	p
MV – RVD	3.52±0.61	3.49±0.54	NS
MV – MLD	1.78±0.93	3.28±0.40	<0.001
MV – acute lumen gain		1.53±0.95	
MV – %DS	51±26	5±11	<0.001
MV lesion length	6.68±5.43		
MB – RVD	3.02±0.51	3.1±0.40	NS
MB – MLD	1.27±0.76	2.96±0.41	<0.001
MB – acute lumen gain		1.69±0.73	
MB – %DS	64±19	3±12	<0.001
MB lesion length	9.95±8.78		
SB – RVD	2.51±0.46	2.52±0.47	NS
SB – MLD	1.29±0.59	1.23±0.74	NS
SB – acute gain		–0.02±0.67	
SB – %DS	48±23	69±21	<0.001
SB – %DS – final		53±25	
SB lesion length	4.37±4.15		

Abbreviations: MV – main vessel, RVD – reference vessel diameter, MLD – minimal lumen diameter, %DS – percent diameter stenosis, MB – main branch, SB – side branch

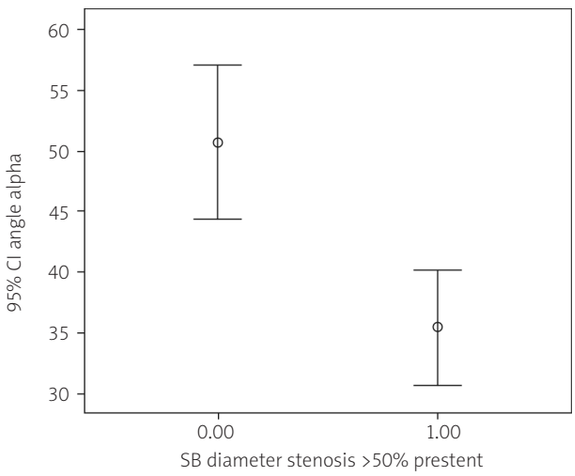

Figure 2. Difference between angle alpha values in groups with and without significant side branch ostial stenosis before main vessel stenting. A cut-off value of 42 degrees could be identified

Follow-up results

Follow-up results are presented in Table IV and Figure 3. There were no deaths during the study period. Four (7%) patients were lost to follow-up. Fifteen (27%) patients were symptomatic and were angiographically followed. In three patients (5%, 3/55) with controversial symptoms (2 females), dobutamine stress test was positive, but coronary angiography did not demonstrate any aggravation of coronary artery disease and no restenosis was detected. Finally, repeated angiography was performed in 31 (56%) patients (Figure 3). In approximately half (14/31) of them angiographic control was motivated by suspicion of target lesion restenosis. The remaining examinations were performed because of suspicion of disease progression in other vessels (11%) or need of revascularisation in a non-target vessel region (20%). The main reason for such indications was the relatively high percentage of multivessel disease in the examined population (47%) (see Table IV).

Although SB restenosis (%DS >70%) was relatively frequent (16/31 – 52% of angiographically followed patients) it was not associated with signs of ischaemia if MV restenosis was missing. In the presented study group, all patients with restenosis in MV (n=8, 15%) always had associated restenosis in SB. By multiple binary logistic analysis independent predictors of SB restenosis were SB lesion length (OR=7.4, 95% CI 1.072-51.741, p=0.042) and reference SB diameter (OR=4.5, 95% CI 1.166-2.10, p=0.048). The model included angle alpha post intervention, SB reference vessel diameter, SB lesion length, sex, SB final minimal lumen diameter, kissing balloon inflation and stent diameter. Interestingly, in ROC curve analysis (with the same parameters as above) the only independent predictor for TLR in SB was angle alpha before stenting (cut-off value 40°, sensitivity 81%, specificity 67%, area under the curve 71%, p=0.05).

The rate of TLR was 26% (8/31 angiographically controlled patients). It was 7% (2/29 lesions) in the drug-eluting stents group and 18% (6/33 lesions) in the BMS group (p=0.05). There were two balloon angioplasties, four drug-eluting stent implantations and two patients were qualified for CABG. The only independent predictors on multivariate binary logistic analysis for TLR were stent type (drug-eluting stent OR 0.072, 95% CI 0.006--0.897, p=0.041) and MB final minimal lumen diameter (OR 0.01, 95% CI 0.000-0.874, p=0.044). The model included MB reference diameter, final minimal lumen diameter in MB after MV stenting, stent type, age, difference between angle A and angle alpha, and performance or not of kissing balloon inflation. In ROC curve analysis independent predictors of TLR were MB minimal lumen diameter at the end of the primary procedure (cut-off value below 2.65 mm, sensitivity 57%, specificity 88%, p=0.029), MB reference diameter (cut-off value below 3.05 mm, sensitivity 86%, specificity 67%, p=0.021) and, surprisingly, angle alpha value post stenting (cut-off value 36°, sensitivity 83%, specificity 77%, p=0.038).

The target vessel failure (TVF) rate was 25% (14/55 patients, the sum of TLR rates and repeat intervention rates in index vessel, but not in target lesion region). In six patients there were additional stent implantations, two proximal and four distal, which extended more than 5 mm of stent edges and as such could not be added to TLR rates. The reasons for TVF were analysed in a logistic model including sex, age, MB reference diameter, stent diameter, stent length, kissing balloon inflation performance, minimal diameter in MB at the end of primary procedure below 2.65 mm and angle alpha value after stenting (Cox & Snell R square =0.500, p=0.004). In this model independent predictors were sex (OR=0.03 for female gender 95% CI 0.002-0.922, p=0.044), MB reference diameter (OR=0.002, 95% CI 0.000-0.722, p=0.038) and stent type (OR=0.026 for DES type 95% CI 0.001-0.686, p=0.029). The cut-off value of MB reference diameter for TVF was determined by multiple ROC analysis (cut-off value below 2.9 mm, sensitivity 64%, specificity 76%, p=0.033).

The cumulative major adverse cardiac events (sum of death, non-fatal myocardial infarction and TVF) rate (34%, 19/55), was almost entirely driven by TVR rates – 25% (n=14) for the total study group. The rate of non-fatal myocardial infarctions was relatively high – 9% (n=5, one Q-wave, four non-Q wave MIs, three of them caused by in-stent restenosis and one by spontaneous plaque rupture in a non-target vessel). The mean time from intervention to myocardial infarction was 6.5 months (range 4-12 months). In one case (with Q-wave myocardial infarction), there was an in-stent thrombosis four months after BMS implantation in the LAD during non-ST elevation myocardial infarction. It is not certain whether the patient was on dual antiplatelet therapy when this complication occurred. All restenosis cases in MV were associated with symptoms and reintervention. Additional progression of coronary disease in the target vessel was detected in five patients (16% of those angiographically followed) and required additional stenting. By logistic regression in a model including sex, age, MB reference diameter, MB final minimal diameter, stent diameter, stent length, stent type (DES vs. BMS), and kissing balloon inflation, only age

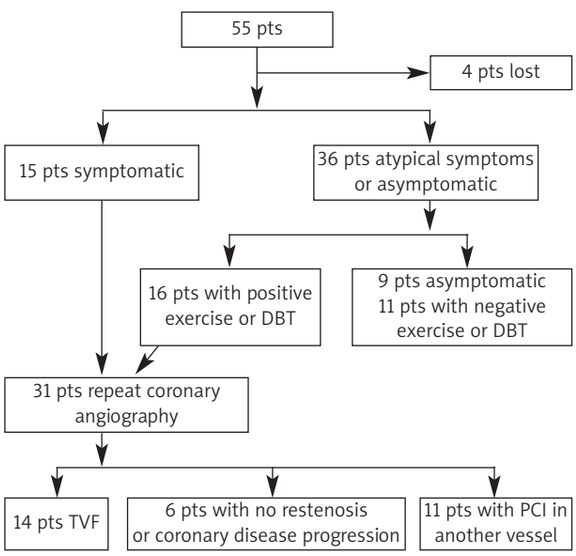

Figure 3. Patient follow-up flow-chart. DBT – dobutamine stress echocardiography test

Table IV. Follow-up results. Data are presented as mean and standard deviations or as absolute values and percentages

Parameter	Result
Time for follow-up [months]	11±3
Myocardial infarction	5 (9%)
Target lesion restenosis MB	8 (15%)
Target lesion restenosis SB	16 (29%)
Time to restenosis [months]	5±5
Target vessel restenosis – proximal	5 (9%)
Target vessel restenosis – distal	5 (9%)
Time to TVR [months]	4±5
Target lesion intervention – TLR	8 (15%)
Target vessel intervention – TVR	6 (11%)
Target vessel failure – TVF	14 (26%)
Interventions in other vessels	11±20
Stent thrombosis	1 (2%)
Time to stent thrombosis [months]	4

was an independent predictor of event rate (OR=1.175, 95% CI 1.005-1.373, p=0.043).

Discussion

The SB compromise (defined as the composite of significant ostial stenosis at lateral vessel ostium and branch vessel closure) was recognised as a significant problem from the early years of interventional cardiology [17-21]. It occurred with rates of 2-38% after balloon angioplasty and around 20% after stent implantation [21]. Although several studies have tried to identify predictors of SB compromise our current knowledge is limited to qualitative predictors of this phenomenon. They can be divided into anatomical and procedural predictors. The most frequently cited anatomical predictors are occurrence of branch vessel from MV stenosis, significant (>50% in most studies) ostial stenosis at SB and small vessel calibre [20, 21, 30-33]. Among procedural predictors the most frequent are high inflation pressure, higher balloon (stent)/artery ratio and balloon predilatation before stent implantation [21, 22, 34-38]. The reason for this uncertainty is lack of direct information about the pathophysiological mechanisms responsible for lateral vessel stenosis or closure. As an unofficial convention the so-called 'plaque shifting' concept was accepted, by analogy with what happened in straight vessel segments [32, 39]. Additional mechanisms were vessel spasm, ostial dissection, stent strut material obstruction of branch vessel lumen and potentially local thrombosis consequently from disturbed flow [20].

We believe that the main mechanism leading to SB compromise is carina displacement from stent struts. This is a predictable phenomenon, as we have shown previously [13-16]. We think that it is the basic mechanism for SB compromise, through which all other mechanisms act. Actually displaced carina leading to predicted % diameter stenosis of 50% in a lesion with SB stenosis of 50% could result in a summation effect and branch closure. Higher implantation pressures and accordingly high stent/artery ratios will cause larger carina displacement and higher ostial stenosis in the branch vessel. The same holds true for the lack of difference in branch compromise depending on implantation technique.

To the best of our knowledge, this is the first study which demonstrates a new predictive factor for main vessel restenosis – angle alpha value before stent implantation. The other predictors of reintervention rates as well as SB restenosis are similar to those already reported in the literature. It is a new finding that angle alpha value is initially associated with higher degree of SB stenosis (see Figure 2), and consequently with in-stent plaque formation during the healing process after index intervention. As such, the geometric milieu of the bifurcation determines not only acute procedure results, but also long-term prognosis of patients. It is notable that similarly to other studies SB restenosis was asymptomatic until significant MB stenosis did not occur. If we take only clinically significant consequences of SB compromise (based on our results),

that is vessel occlusion procedurally and MV restenosis, resulting in the revascularisation procedure, we can speculate that bifurcation lesions could be divided into two types depending on angle alpha value. The cut-off value of 40° could be accepted as a combination of cut-off values for branch occlusion and MB revascularisation. This could be a basis for a simple classification, which may predict event rates in coronary bifurcation intervention.

The results of our study once more confirm the advantage of drug-eluting stents over bare metal stents in bifurcation lesion treatment. Drug-eluting stents reduce rates of restenosis and in our group do not influence rates of reinfarction.

The single case of in-stent thrombosis is difficult to comment on, as that was in a patient with bare metal stent implantation during primary angioplasty, four months after the initial procedure. Bifurcation lesion *per se* predisposes to higher thrombosis risk [40, 41] and combination with previous plaque destabilisation could increase the risk of adverse outcome.

The main limitation of our study is its retrospective nature and inability to follow 100% of patients. Moreover, a significant number of patients with bifurcation lesions treated had angiograms of insufficient quality to permit accurate geometric analysis. We believe that with the introduction of computer tomography giving a real three-dimensional view of coronary anatomy and exact determination of vessel angulations this problem will be overcome.

Conclusions

Our study demonstrates the influence of geometric factors, mainly parent vessel – side branch angle, on SB compromise during intervention and at long term. The proposed mechanism is carina displacement. Thus, angle alpha predicts SB compromise after MV stenting and influences restenosis in MV in long term.

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