Journal of the American Heart Association

ORIGINAL RESEARCH

# Clinical Outcomes of Percutaneous Coronary Intervention for Bifurcation Lesions According to Medina Classification

Mohamed O. Mohamed, PhD; Pablo Lamellas, MD; Ariel Roguin, PhD; Rohit M. Oemrawsingh, PhD; Alexander J. J. Ijsselmuiden, PhD; Helen Routledge, MD; Frank van Leeuwen, MD; Roxane Debrus, MSc; Marco Roffi, PhD; Mamas A. Mamas, DPhil; on behalf of the e-Ultimaster investigators*

**BACKGROUND:** Coronary bifurcation lesions (CBLs) are frequently encountered in clinical practice and are associated with worse outcomes after percutaneous coronary intervention. However, there are limited data around the prognostic impact of different CBL distributions.

**METHODS AND RESULTS:** All CBL percutaneous coronary intervention procedures from the prospective e-Ultimaster (Prospective, Single-Arm, Multi Centre Observations Ultimaster Des Registry) multicenter international registry were analyzed according to CBL distribution as defined by the Medina classification. Cox proportional hazards models were used to compare the hazard ratio (HR) of the primary outcome, 1-year target lesion failure (composite of cardiac death, target vessel–related myocardial infarction, and clinically driven target lesion revascularization), and its individual components between Medina subtypes using Medina 1.0.0 as the reference category. A total of 4003 CBL procedures were included. The most prevalent Medina subtypes were 1.1.1 (35.5%) and 1.1.0 (26.8%), whereas the least prevalent was 0.0.1 (3.5%). Overall, there were no significant differences in patient and procedural characteristics among Medina subtypes. Only Medina 1.1.1 and 0.0.1 subtypes were associated with increased target lesion failure (HR, 2.6 [95% CI, 1.3–5.5] and HR, 4.0 [95% CI, 1.6–9.0], respectively) at 1 year, compared with Medina 1.0.0, prompted by clinically driven target lesion revascularization (HR, 3.1 [95% CI, 1.1–8.6] and HR, 4.6 [95% CI, 1.3–16.0], respectively) as well as cardiac death in Medina 0.0.1 (HR, 4.7 [95% CI, 1.0–21.6]). No differences in secondary outcomes were observed between Medina subtypes.

**CONCLUSIONS:** In a large multicenter registry analysis of coronary bifurcation percutaneous coronary intervention procedures, we demonstrate prognostic differences in 1-year outcomes between different CBL distributions, with Medina 1.1.1 and 0.0.1 subtypes associated with an increased risk of target lesion failure.

**Key Words:** bifurcation ■ drug-eluting stent ■ Medina classification ■ outcomes ■ percutaneous coronary intervention



Coronary bifurcation lesions (CBLs) are some of the most challenging and frequently encountered lesion subsets in interventional practice, representing nearly 20% of all percutaneous coronary intervention (PCI) procedures.[1,2] Although several classifications of CBLs exist, the Medina classification, endorsed by major bodies such as the European Bifurcation Club, is the most widely used.[2–5] This classification assigns a binary value (0 or 1) to the proximal and distal main branches (MBs) as well as the side branch (SB), in that respective order, based on the presence (1) or absence (0) of significant plaque burden (≥50% stenosis) in that vascular segment.[4] Furthermore, CBLs can be classified into true bifurcation lesions, if both the MB and SB have

Correspondence to: Mamas A. Mamas, DPhil, Keele Cardiovascular Research Group, Centre for Prognosis Research, Institute for Primary Care and Health Sciences, Keele University, Keele, United Kingdom. Email: mamasmamas1@yahoo.co.uk

*A complete list of the e-Ultimaster investigators can be found in the Supplemental Material.

Supplemental Material is available at https://www.ahajournals.org/doi/suppl/10.1161/JAHA.122.025459

For Sources of Funding and Disclosures, see page 9.



*JAHA* is available at: www.ahajournals.org/journal/jaha

## CLINICAL PERSPECTIVE

### What Is New?

- This is the first study to examine the prognostic difference in 1-year clinical outcomes after coronary bifurcation lesion percutaneous coronary intervention according to Medina subtype in a large multinational cohort.
- Our findings demonstrate that the most prevalent coronary bifurcation lesion subtypes are Medina 1.1.1 (35.5%) and 1.1.0 (26.8%), whereas the least prevalent is Medina 0.0.1 (3.5%).
- Only 2 Medina subtypes (1.1.1 and 0.0.1) were associated with increased target lesion failure at 1 year, whereas no prognostic differences were observed for other subtypes.

### What Are the Clinical Implications?

- Greater caution is warranted for patients with specific coronary bifurcation lesion distributions, namely Medina 1.1.1 and 0.0.1, who may need to be followed up more closely given their increased risk of target lesion failure at 1 year following intervention.
- Further research is needed to further optimize outcomes of patients with isolated side branch lesions (Medina 0.0.1) who are at a 4-fold higher risk of target lesion failure at 1 year.

### Nonstandard Abbreviations and Acronyms

| | |
|---|---|
| **CBL** | coronary bifurcation lesion |
| **MB** | main branch |
| **SB** | side branch |
| **TLF** | target lesion failure |
| **TLR** | target lesion revascularization |
| **TVF** | target vessel failure |

significant stenosis, and nontrue bifurcation lesions if either the MB or SB is not significantly stenosed.

Although previous studies have examined clinical outcomes of CBL PCI according to lesion complexity (simple versus complex bifurcations) or true versus nontrue bifurcations,[6–9] none have systematically examined the prognostic impact of disease distribution within the CBL on mid- or long-term outcomes following PCI.[10–12]

The present study sought to compare the impact of disease distribution according to Medina classification on 1-year clinical outcomes adjudicated by an independent clinical events committee among patients undergoing bifurcation PCI within the e-Ultimaster (Prospective, Single-Arm, Multi Centre Observations Ultimaster Des Registry).

## METHODS

### Study Data Set

The e-Ultimaster study is a large, international, prospective observational study that enrolled 37 198 patients between October 2014 and June 2018 in 378 hospitals from 50 countries including sites in Europe, Asia, Africa, the Middle East, South America, and Mexico. Eligibility criteria were minimal to enroll an all-comer population, and included (1) age ≥18 years and (2) an indication for a PCI according to routine hospital practice. The Ultimaster sirolimus-eluting coronary stent (Terumo, Tokyo, Japan) is made of cobalt-chromium with a strut thickness of 80 μm. Sirolimus is released from an abluminal applied bioresorbable polymer coating (poly-D,L-lactic acid polycaprolactone) that is fully metabolized through dl-lactide and caprolactone into carbon dioxide and water in 3 to 4 months. The study was approved by the ethical committees of the participating sites, and all patients provided written informed consent. The clinicaltrial.gov identifier is NCT02188355. The data used for the purpose of this study are only available to designated researchers and cannot be shared with other researchers. However, all efforts were made to describe the methods in detail.

### Study Population and Follow-Up

All patients with a single CBL at the index procedure from the e-Ultimaster study were included, stratified into 7 permutations according to their Medina classification (true bifurcations: 0.1.1, 1.0.1, 1.1.1; nontrue bifurcations: 0.0.1, 0.1.0, 1.0.0, 1.1.0) (Figure S1). There were no restrictions on the clinical indication, the number of lesions to be treated, or number of stents to be implanted. Because the Medina classification was not available for 56 (1.4%) patients, they were excluded from our analysis. Follow-up was up to 1 year, except

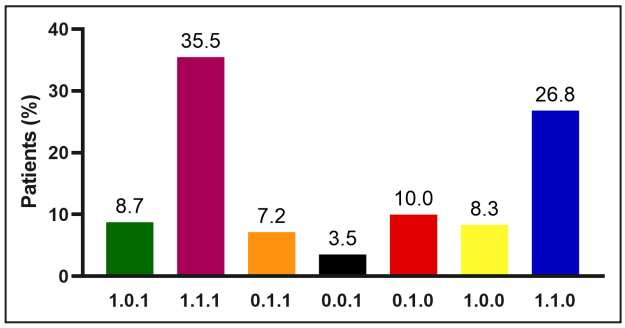


**Figure 1. Distribution of coronary bifurcation lesions as per the Medina classification subtype.**

**Table 1. Baseline Patient and Procedural Characteristics According to Medina Classification Subtype**

| Characteristic | True bifurcation | | | Nontrue bifurcation | | | | |
|---|---|---|---|---|---|---|---|---|
| | Medina 1.0.1 | Medina 1.1.1 | Medina 0.1.1 | Medina 0.0.1 | Medina 0.1.0 | Medina 1.0.0 | Medina 1.1.0 | *P* value |
| | n=356 | n=1420 | n=291 | n=137 | n=399 | n=336 | n=1064 | |
| Age, y, mean±SD [n] | 65.1±11.3 [356] | 65.7±11.4 [1420] | 64.9±10.7 [291] | 65.9±10.8 [137] | 65.2±11.2 [399] | 65.0±11.6 [336] | 65.8±10.8 [1064] | 0.709 |
| Men, % [n] | 73.3 [261/356] | 77.5 [1101/1420] | 74.2 [216/291] | 70.8 [97/137] | 75.9 [303/399] | 77.1 [259/336] | 77.4 [824/1064] | 0.338 |
| Diabetes, % [n] | 25.1 [89/354] | 28.7 [405/1410] | 29.3 [84/287] | 31.4 [43/137] | 26.6 [106/399] | 21.9 [73/334] | 25.6 [271/1057] | 0.111 |
| Insulin-dependent diabetes, % [n] | 4.8 [17/354] | 5.3 [74/1410] | 8.0 [23/287] | 5.8 [8/137] | 4.5 [18/399] | 4.2 [14/334] | 4.9 [52/1057] | 0.412 |
| Hypertension, % [n] | 68.7 [233/339] | 69.1 [9400] | 71.4 [200/280] | 72.9 [97/133] | 71.1 [273/384] | 66.3 [212] | 67.5 [674/998] | 0.588 |
| Hypercholesterolemia, % [n] | 57.9 [188/325] | 62.2 [825/1326] | 61.3 [171/279] | 58.9 [76/129] | 63.9 [246] | 61.5 [193/314] | 63.8 [629/986] | 0.555 |
| Current smoking, % [n] | 23.7 [70/295] | 23.7 [288/1215] | 19.6 [49/250] | 24.8 [27/109] | 23.1 [80/347] | 27.3 [76/278] | 23.6 [211/894] | 0.608 |
| Previous MI, % [n] | 24.3 [83/341] | 25.4 [3479] | 26.3 [75/285] | 27.3 [36/132] | 21.0 [82/391] | 20.7 [67/324] | 23.5 [246/1046] | 0.318 |
| Previous PTCA, % [n] | 26.5 [93/351] | 29.5 [410/1392] | 30.3 [87/287] | 38.2 [52] | 30.9 [123/398] | 28.7 [94/328] | 32.0 [337/1052] | 0.179 |
| Previous CABG, % [n] | 5.2 [18/347] | 5.4 [75/1383] | 4.5 [13/287] | 3.7 [5] | 2.8 [11/394] | 2.7 [9/329] | 4.4 [46/1046] | 0.210 |
| Chronic coronary syndrome, % [n] | 48.0 [171/356] | 49.0 [696/1420] | 55.9 [162/290] | 49.6 [68/137] | 55.8 [222/398] | 49.4 [166/336] | 54.6 [580/1063] | 0.020 |
| Acute coronary syndrome, % [n] | 52.0 [185/356] | 51.0 [724/1420] | 44.1 [128/290] | 50.4 [69/137] | 44.2 [176/398] | 50.6 [170/336] | 45.4 [483/1063] | 0.020 |
| Unstable angina | 13.8 [49/356] | 13.3 [189/1420] | 10.7 [31/290] | 23.4 [32/137] | 13.1 [52/398] | 9.8 [33/336] | 11.7 [124/1063] | 0.004 |
| NSTEMI | 24.7 [88/356] | 23.0 [327/1420] | 24.1 [70/290] | 16.1 [22/137] | 20.6 [82/398] | 25.9 [87/336] | 21.9 [233/1063] | 0.237 |
| STEMI | 13.5 [48/356] | 14.7 [208/1420] | 9.3 [27/290] | 11.0 [15/137] | 10.6 [42/398] | 14.9 [50/336] | 11.9 [126/1063] | 0.064 |
| Femoral access, % [n] | 21.1 [75/356] | 21.3 [302/1420] | 19.9 [58/291] | 18.3 [25/137] | 15.8 [63/399] | 15.5 [52/336] | 17.9 [190/1064] | 0.059 |
| Radial access, % [n] | 79.5 [283/356] | 79.4 [1127/1420] | 80.8 [235/291] | 81.0 [111/137] | 85.2 [340/399] | 83.9 [282/336] | 83.5 [888/1064] | 0.042 |
| Intracoronary imaging use, % [n] | 7.6 [27/356] | 10.4 [148/1420] | 10.7 [31/291] | 11.0 [15/137] | 12.8 [51/399] | 13.1 [44/336] | 14.5 [154/1064] | 0.008 |
| Lesion preparation, % [n] | | | | | | | | |
| Atherectomy | 0.6 [2/356] | 2.0 [29/1420] | 0.0 [0/291] | 0.0 [0/137] | 1.0 [4/399] | 0.6 [2/336] | 1.5 [16/1064] | 0.020 |
| Cutting balloon | 0.8 [3/356] | 1.6 [23/1420] | 3.4 [10/291] | 1.5 [2/137] | 1.5 [6/399] | 1.2 [4/336] | 1.3 [14/1064] | 0.190 |
| Thrombus aspiration | 3.1 [11/356] | 2.8 [40/1420] | 1.7 [5/291] | 1.5 [2/137] | 3.0 [12/399] | 3.9 [13/336] | 2.8 [30/1064] | 0.720 |
| Overall no. of lesions treated, mean±SD [N] | 1.5±0.7 [356] | 1.4±0.7 [1420] | 1.5±0.8 [291] | 1.4±0.8 [137] | 1.3±0.6 [399] | 1.4±0.6 [336] | 1.4±0.7 [1063] | 0.058 |
| No. of stents implanted, mean±SD [N] | 1.8±1.0 [356] | 2.0±1.1 [1420] | 2.1±1.1 [291] | 1.7±0.9 [137] | 1.5±0.8 [399] | 1.5±0.8 [336] | 1.8±1.0 [1063] | <0.001 |

(*Continued*)

**Table 1. Continued**

| Characteristic | True bifurcation | | | Nontrue bifurcation | | | | |
|---|---|---|---|---|---|---|---|---|
| | Medina 1.0.1 | Medina 1.1.1 | Medina 0.1.1 | Medina 0.0.1 | Medina 0.1.0 | Medina 1.0.0 | Medina 1.1.0 | *P* value |
| | n=356 | n=1420 | n=291 | n=137 | n=399 | n=336 | n=1064 | |
| Bifurcation main vessel, % [n] | | | | | | | | |
| Left main | 5.9 [21/356] | 12.8 [181/1420] | 8.6 [25/291] | 2.2 [3/137] | 8.3 [33/399] | 11.9 [40/336] | 14.9 [158/1064] | <0.001 |
| Left anterior descending | 53.4 [190/356] | 63.2 [897/1420] | 69.1 [201/291] | 47.5 [65/137] | 65.4 [261/399] | 53.3 [179/336] | 58.1 [618/1064] | <0.001 |
| Left circumflex | 34.8 [124/356] | 17.0 [241/1420] | 18.2 [53/291] | 43.1 [59/137] | 19.3 [77/399] | 22.3 [75/336] | 17.3 [184/1064] | <0.001 |
| Right coronary artery | 5.9 [21/356] | 7.0 [100/1420] | 4.1 [12/291] | 7.3 [10/137] | 7.0 [28/399] | 12.5 [42/336] | 9.8 [104/1064] | <0.001 |
| Bifurcation technique, % [n] | | | | | | | | |
| 1 stent | 64.6 [212/328] | 65.2 [897/1376] | 52.2 [145/278] | 56.8 [21/37] | 97.4 [378/388] | 96.3 [316/328] | 94.3 [987/1047] | <0.001 |
| 2 stents | 35.4 [116/328] | 34.8 [479/1376] | 47.8 [133/278] | 43.2 [16/37] | 2.6 [10/388] | 3.7 [12/328] | 5.7 [60/1047] | <0.001 |
| POT | 29.2 [103/353] | 41.1 [581/1415] | 31.5 [91/289] | 15.3 [21/137] | 38.9 [155/398] | 26.6 [89/335] | 30.9 [322/1042] | <0.001 |

CABG indicates coronary artery bypass graft surgery; MI, myocardial infarction; NSTEMI, non–ST-segment–elevation myocardial infarction; POT, proximal optimization technique; PTCA, percutaneous transluminal coronary angiography; and STEMI, ST-segment–elevation myocardial infarction.

for patients in whom no Ultimaster stent was implanted, in which follow-up was only available to discharge. The event rates (see Outcomes) at 1-year follow-up were calculated based upon the number of patients who had a 1-year follow-up visit or experienced a clinical event. All primary outcome-related adverse events were adjudicated by an independent clinical events committee.

## Outcomes

The primary outcome measure was target lesion failure (TLF), defined as the composite of cardiac death, target vessel–related myocardial infarction, and clinically driven target lesion revascularization. Secondary outcomes included target vessel failure (TVF), composite of cardiac death, target vessel myocardial infarction and clinically driven target vessel revascularization, and patient-oriented composite end point, defined as a composite of all-cause death, any myocardial infarction (MI), and any revascularization. For MI, the extended historical definition was applied that primarily uses creatine kinase myocardial band, or if not available troponin, as a cardiac biomarker criterion.[13] All deaths, MI, target lesion revascularizations (TLRs) or target vessel revascularizations, and stent thrombosis were adjudicated by an independent clinical events committee.

## Statistical Analysis

Continuous variables are reported as mean along with SD and compared using the ANOVA test, whereas categorial variables are reported as frequency and percentage and compared using the $\chi^2$ test. Analyses were performed with SAS version 9.4 software (SAS Institute, Cary, NC). Cox proportional hazards models were performed using Medina 1.0.0 as the reference category to assess the hazard ratio (HR) of 1-year outcomes across different Medina subtypes, adjusting for PCI indication (chronic coronary syndrome, unstable angina, non–ST-segment–elevation myocardial infarction, and ST-segment–elevation myocardial infarction), CBL vessel location (right coronary artery, left main stem, left anterior descending artery, and left circumflex artery), radial access, stenting strategy (1 versus 2 stents), and use of intracoronary imaging and/or proximal optimization technique.

# RESULTS

A total of 4003 patients undergoing PCI for CBLs at the index procedure were included in the analysis. The most prevalent lesion subtypes were Medina 1.1.1 (n=1420, 35.5%) and Medina 1.1.0 (n=1064, 26.8%), whereas the least prevalent subtype was Medina 0.0.1 (n=137, 3.5%) (Figure 1).

**Table 2. One-Year Clinical Outcomes According to Medina Classification Subtype**

| | True bifurcation | | | Nontrue bifurcation | | | | |
|---|---|---|---|---|---|---|---|---|
| | 1.0.1 | 1.1.1 | 0.1.1 | 0.0.1 | 0.1.0 | 1.0.0 | 1.1.0 | |
| Outcome | n=337 | n=1368 | n=276 | n=136 | n=385 | n=320 | n=1033 | *P* value |
| TLF, % [n] | 4.5% [15] | 6.1% [83]* | 2.9% [8] | 8.1% [11]† | 3.4% [13] | 2.5% [8] | 4.7% [49] | 0.016 |
| TVF, % [n] | 5.3% [18] | 7.4% [101]* | 2.9% [8] | 8.8% [12]* | 3.9% [15] | 4.1% [13] | 5.8% [60] | 0.009 |
| POCE, % [n] | 9.8% [33] | 10.8% [148]* | 6.2% [17] | 9.6% [13] | 7.0% [27] | 6.3% [20] | 9.7% [100] | 0.040 |
| All-cause death, % [n] | 3.6% [12] | 3.6% [49] | 0.7% [2] | 2.9% [4] | 2.9% [11] | 1.9% [6] | 1.9% [20] | 0.056 |
| Cardiac death, % [n] | 1.2% [4] | 2.4% [33] | 0.4% [1] | 2.9% [4] | 2.1% [8] | 0.9% [3] | 1.5% [15] | 0.115 |
| TV-MI, % [n] | 1.5% [5] | 1.8% [25] | 0.4% [1] | 0.7% [1] | 0.3% [1] | 0.9% [3] | 1.4% [14] | 0.179 |
| CD-TVR, % [n] | 3.9% [13] | 4.9% [67] | 2.5% [7] | 6.6% [9] | 1.6% [6] | 2.8% [9] | 4.1% [42] | 0.028 |
| CD-TLR, % [n] | 3.0% [10] | 3.4% [46]* | 2.5% [7] | 5.2% [7]* | 1.0% [4] | 1.3% [4] | 2.8% [29] | 0.068 |
| Definite/probable ST, % [n] | 1.2% [4] | 1.1% [15] | 0.0% [0] | 0.0% [0] | 0.3% [1] | 0.9% [3] | 0.7% [7] | 0.290 |

CD-TLR indicates clinically driven target lesion revascularization; CD-TVR, clinically driven target vessel revascularization; POCE, patient-oriented composite end point (composite of all-cause death, any myocardial infarction, and any revascularization); ST, stent thrombosis; TLF, target lesion failure (composite of cardiac death, target vessel myocardial infarction, and clinically driven target lesion revascularization); TVF, target vessel failure (composite of cardiac death, target vessel myocardial infarction, and clinically driven target vessel revascularization); and TV-MI, target vessel myocardial infarction.

*$P$<0.05 vs Medina 1.0.0.

†$P$<0.01 vs Medina 1.0.0.

## Patient and Procedural Characteristics

The median age for the entire cohort was ≈65 years, with the majority being men (70.8%–77.5%), undergoing PCI for chronic coronary syndrome (48.0%–55.9%) (Table 1). However, no differences were observed between Medina subtypes for characteristics such as age, sex, diabetes, hypertension, hypercholesterolemia and previous MI, coronary artery bypass graft surgery, and percutaneous transluminal coronary angioplasty. Acute coronary syndrome was the most frequent indication for Medina 1.0.1 (52.0%) and the least frequent for Medina 0.1.1 (44.1%).

In terms of procedural characteristics, the rate of use of intracoronary imaging was highest in the Medina 1.1.0 subtype (14.5%) and lowest in the Medina 1.0.1 subtype (7.6%) (Table 1). Although there was no difference in the mean number of total lesions treated among Medina subtypes, the greatest number of stents implanted were in Medina 0.1.1 (2.1±1.1), whereas the lowest was in Medina 0.1.0 and 1.0.0 (1.5±0.8 each). Overall, the left anterior descending artery was the most treated vessel, and this was most frequent in Medina 0.1.1 (69.1%) and least frequent in Medina 0.0.1 (47.5%). The majority of CBLs were treated by a

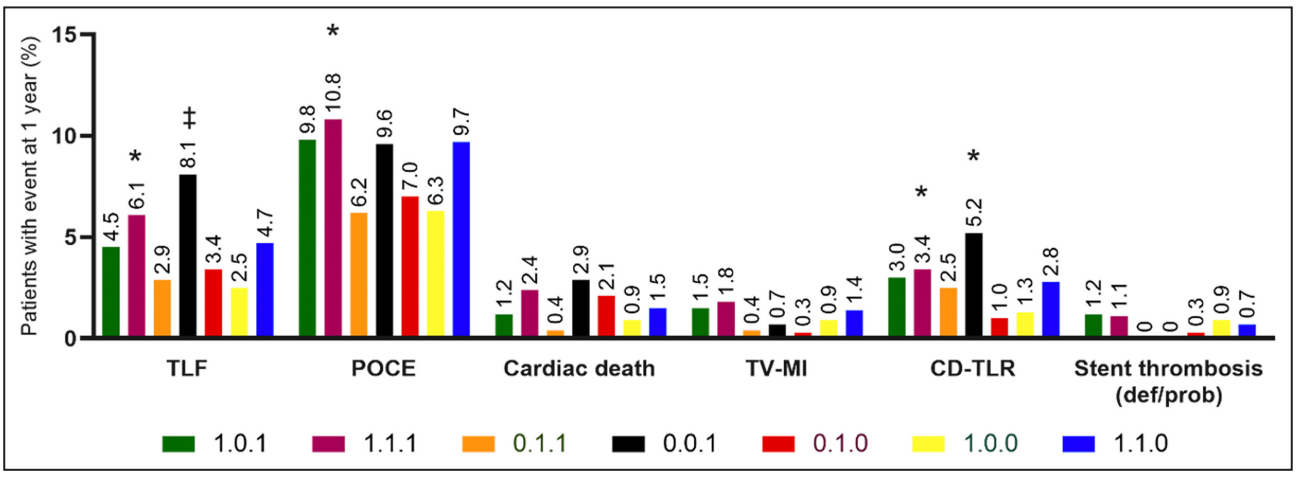


**Figure 2. Clinical outcomes at 1 year as per the Medina classification subtype.**
CD-TLR indicates clinically driven target lesion revascularization; def/prob, definite/probable; POCE, patient-oriented composite end point (composite of all-cause death, any myocardial infarction, and any revascularization); TLF, target lesion failure (composite of cardiac death, target-vessel myocardial infarction, and clinically driven target lesion revascularization); and TV-MI, target vessel myocardial infarction. Symbols indicate significant $P$ value vs Medina 1.0.0: *$P$<0.05 and ‡$P$<0.01, the rest are nonsignificant ($P$>0.05).

**Table 3. HR With 95% CI of Clinical Outcomes at 1 Year According to Medina Classification***

| Outcome | HR (95% CI) | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | True bifurcation | | | | | | Nontrue bifurcation | | | | | |
| | 1.0.1 | *P* value | 1.1.1 | *P* value | 0.1.1 | *P* value | 0.0.1 | *P* value | 0.1.0 | *P* value | 1.1.0 | *P* value |
| Target lesion failure | 2.0 (0.9–4.9) | 0.106 | 2.6 (1.3–5.5) | 0.001 | 1.3 (0.5–3.5) | 0.629 | 4.0 (1.6–9.9) | 0.003 | 1.5 (0.6–3.7) | 0.332 | 1.9 (0.9–4.1) | 0.083 |
| Cardiac death | 1.6 (0.4–7.2) | 0.547 | 2.9 (0.9–9.6) | 0.086 | 0.5 (0.0–4.7) | 0.534 | 4.7 (1.0–21.6) | 0.044 | 2.7 (0.7–10.1) | 0.148 | 1.6 (0.5–5.4) | 0.474 |
| Target vessel myocardial infarction | 1.7 (0.4–7.1) | 0.490 | 2.2 (0.6–7.4) | 0.211 | 0.4 (0.0–3.7) | 0.406 | 0.9 (0.1–8.6) | 0.918 | 0.3 (0.0–3.1) | 0.322 | 1.5 (0.4–5.3) | 0.512 |
| Clinically driven target lesion revascularization | 2.8 (0.9–8.9) | 0.089 | 3.1 (1.1–8.6) | 0.034 | 2.3 (0.7–7.9) | 0.197 | 4.6 (1.3–16.0) | 0.015 | 0.9 (0.2–3.8) | 0.926 | 2.3 (0.8–6.5) | 0.120 |
| Stent thrombosis (definite/probable) | 1.0 (0.2–4.7) | 0.988 | 1.1 (0.3–4.0) | 0.877 | † | † | † | † | 0.3 (0.0–3.2) | 0.335 | 0.8 (0.2–3.1) | 0.738 |

HR indicates hazard ratio.
*Reference is Medina 1.0.0.
†Zero events.

1-stent technique, especially the nontrue bifurcation subtypes Medina 0.1.0 (97.4%), Medina 1.0.0 (96.3%), and Medina 1.1.0 (94.3%), whereas the use of a 2-stent technique was highest in true bifurcations (34.8%–47.8%) as well as Medina 0.0.1 (43.2%). The proximal optimization technique was more frequently used in the Medina 1.1.1 subtype (41.1%) and least commonly in Medina 0.0.1 (15.3%). Furthermore, the overall use of the proximal optimization technique and poststenting kissing balloon was higher in 2-stent than 1-stent techniques (77.4% versus 31.1% and 49.4% versus 36.8%, respectively). Several 2-stent techniques were used for true and nontrue bifurcations (Tables S1 and S2, respectively), T-stenting (23.8%) being the most used, followed by the Crush technique (17.0%) and TAP (16.1%) in true bifurcation lesions. There was a variation depending on the type of the true bifurcation. For nontrue bifurcations, another technique that was not specified was the most used (29.6%), whereas for true bifurcations, T-stenting was the common strategy (23.8%).

## Clinical Outcomes

Overall, TLF and TVF rates were highest in Medina 1.1.1 (6.1% and 7.4%, respectively) and Medina 0.0.1 (8.1% and 8.8%, respectively) subtypes, whereas the lowest TLF and target vessel revascularization rates were in the Medina 1.0.0 and Medina 0.1.1 subtypes, respectively (TLF: 2.5%, TVF: 2.9%) (Table 2, Figure 2). The high TLF and TVF rates were primarily driven by high rates of clinically driven TLR and target vessel revascularization, respectively. Patient-oriented composite end point rates were highest among Medina 1.1.1 (10.8%), Medina 0.0.1 (9.6%), and Medina 1.1.0 (9.7%) subtypes compared with all other bifurcation distributions. No statistically significant differences between Medina subtypes were observed for other outcomes including all-cause and cardiac death, target vessel MI, and definite/probable stent thrombosis.

Using Medina 1.0.0 as the reference category, the risk of 1-year TLF was only increased in Medina 1.1.1 (HR, 2.6 [95% CI, 1.3–5.5]) and Medina 0.0.1 (HR, 4.0 [95% CI, 1.6–9.9]) subtypes, driven by an increased risk of clinically driven TLR in either subtype (Medina 1.1.1: HR, 3.1 [95% CI, 1.1–8.6]; Medina 0.0.1: HR, 4.6 [95% CI, 1.3–16.0]) and cardiac death in the Medina 0.0.1 subtype (HR, 4.7 [95% CI, 1.0–21.6]) (Table 3, Figure 3). No other differences in risk of adverse clinical outcomes (target vessel MI, cardiac death, and stent thrombosis) were observed between Medina 1.0.0 and other Medina subtypes.

## Subgroup Analysis

Comparison by Medina subtype found no difference between stenting techniques (1 versus 2 stents) except

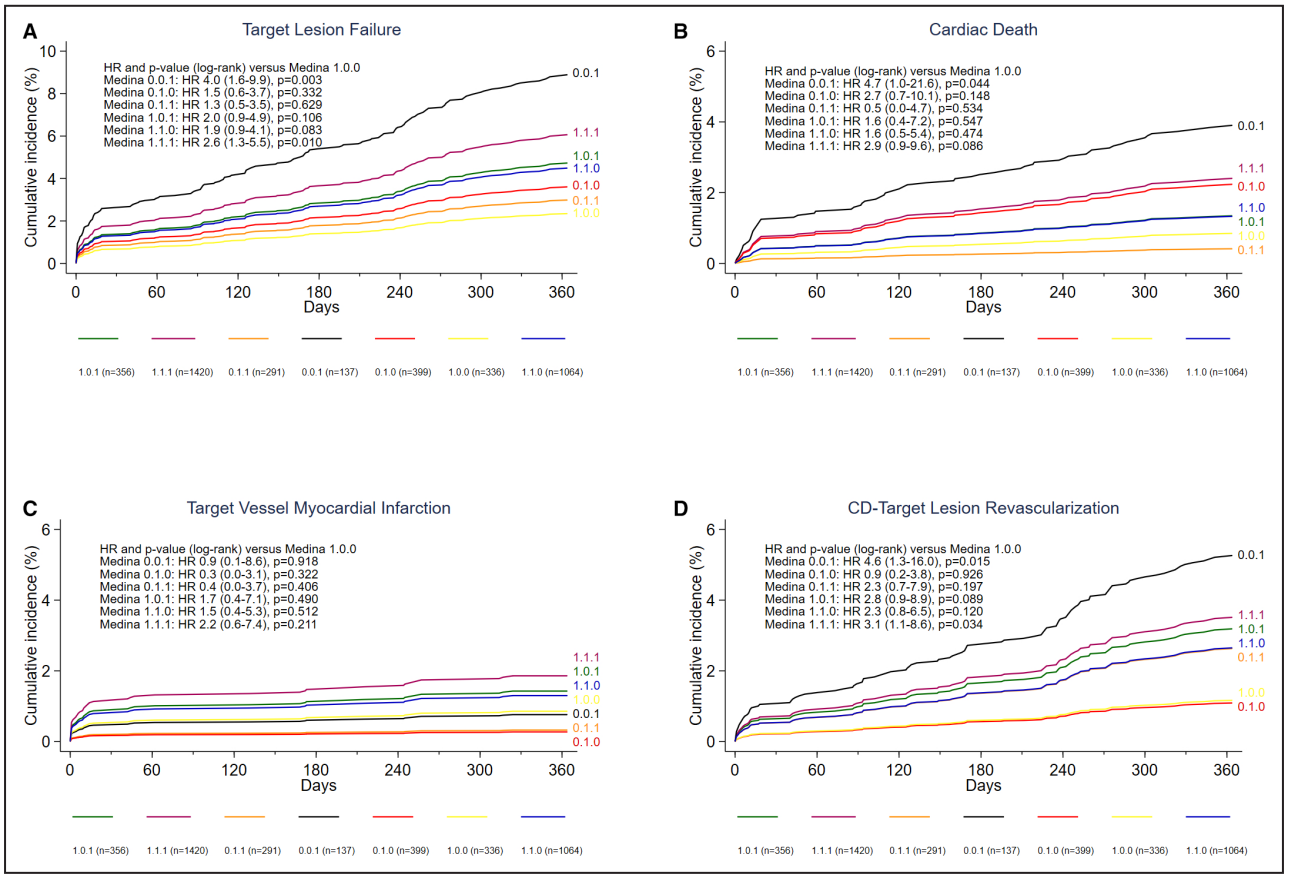


**Figure 3. Hazard ratio (HR) and 95% CI for individual Medina subtypes for (A) target lesion failure, (B) cardiac death, (C) target vessel myocardial infarction, and (D) clinically driven (CD) target lesion revascularization.**
Reference is Medina 1.0.0.

in the Medina 1.0.0 group, where a 2-stent strategy was associated with higher rates of clinically driven TLR (8.3% versus 1.0%, $P<0.05$) (Table S3). Furthermore, there were no differences in clinical outcomes at 1 year per type of 2-stent technique except for all-cause death, with the lowest rate observed with V-stenting (0.0%, 0/48) and the highest with the Culotte technique (9.2%, 8/87). (Table S4).

Tables S5 and S6 summarize 1-year clinical outcomes for bifurcation lesions involving the left main and left anterior descending arteries, respectively, as per the Medina classification. No differences in clinical outcomes were observed between Medina subtypes, except for patient-oriented composite end point for lesions involving the left main artery for which the highest rate was observed in Medina 1.1.1 (21.9%) and the lowest in Medina 0.0.1 (0.0%).

## DISCUSSION

The present study is the first to outline the impact of lesion distribution according to Medina classification on 1-year clinical outcomes among >4000 patients undergoing CBL PCI using the same contemporary stent platform. Our findings can be summarized as follows (Figure 4). First, we found that Medina 1.1.1 (35.5%) and Medina 1.1.0 (26.8%) were the most prevalent CBL distributions, whereas Medina 0.0.1 (3.5%) was the least prevalent. Second, no clinically meaningful differences in patient characteristics were observed following stratification for Medina subtypes. Despite this, PCI of Medina 1.1.1 and 0.0.1 subtypes was associated with significantly higher crude rates of TLF compared with all other bifurcation distributions and were independently associated with an increased hazard of TLF (2.6- and 4-fold, respectively) at 1 year.

Although previous studies have examined differences in procedural outcomes between true and non-true CBLs, there are limited data on the prognostic impact of disease distribution in CBLs.[10–12] Our findings highlight differences in 1-year outcomes between CBL distributions, with the greatest hazard for TLF and TVF observed among Medina 1.1.1 and Medina 0.0.1, even after adjustment for baseline differences between Medina subtypes. Given the limited previous literature comparing outcomes between CBL lesion

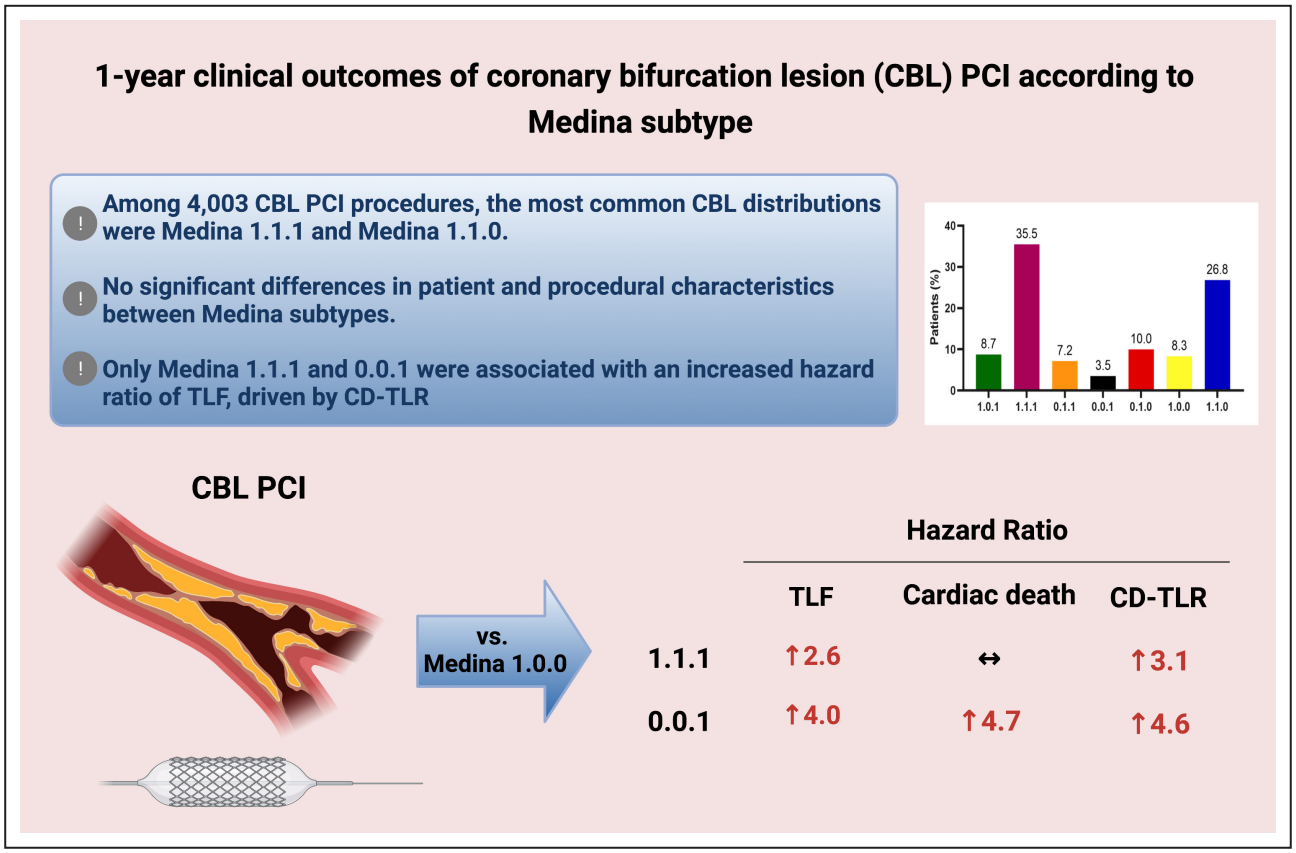


**Figure 4. Summary of study findings.**
CBL indicates coronary bifurcation lesion; CD-TLR, target lesion revascularization; PCI, percutaneous coronary intervention; and TLF, target lesion failure.

distributions, it is difficult to place our findings within the context of other studies. A study of 2897 patients undergoing CBL PCI reported higher crude rates of Major acute cardiovascular events and cardiac death/MI among specific Medina subtypes (Medina 1.1.1: 12.4% and 4.5%, respectively; Medina 0.1.1: 13.9% and 3.7%, respectively).[10] However, the authors did not compare outcomes between different Medina subtypes except within true CBL groups (Medina 1.1.1 and Medina 0.1.1 versus Medina 1.0.1) and reported no differences between these groups. Furthermore, their findings were derived from an old procedural cohort (enrolled between 2003 and 2009), managed with older (first and second) generation drug-eluting stents. In contrast, Todara et al reported no difference in in-hospital and 12-month cardiac death, TLR, and reinfarction between Medina 1.1.1 and all other subtypes combined.[11] However, their analysis was based on a small sample size (n=120, including n=25 with Medina 1.1.1) from an old procedural cohort (2005–2006), where 25% of patient were not managed with a drug-eluting stent.

The finding that 0.0.1 isolated SB disease has the highest hazard of TLF is interesting and deserves further comment. Our subgroup analysis suggests that the worse outcomes associated with 0.0.1 are observed irrespective of whether a single- or 2-stent approach is used. Isolated SB disease is complex to treat, because lesions are often fibrocalcific with significant recoil, which makes achieving good minimal luminal area challenging, particularly if the lesions are not adequately prepared.[14,15] Furthermore, isolated SB lesions often supply a limited myocardial territory (<10%), with only 1 in 5 non–left main artery SB lesions shown to supply >10% fractional myocardial mass in a multicenter registry analysis of 482 patients undergoing computed tomography coronary angiography.[16] Isolated SB lesions are also difficult to treat, because identification of the ostium may be challenging, resulting in geographical or ostial miss, or compromise of the main vessel, which may account for 27% of isolated SB lesions treated with a 2-stent approach. Furthermore, stent underexpansion or recoil, smaller SB vessel size in relation to the MB, and SB length may all contribute to the worse outcomes observed in this group.[17–19] The mean stent diameter for SB 0.0.1 lesions in our study was 2.79 mm compared with >3 mm used to treat MB lesions. Consequently, many operators may choose to treat these lesions conservatively or use alternative interventional strategies such as drug-eluting balloons, although there is limited evidence to support the latter.

An observational study of 49 patients of Medina 0.0.1 lesions with associated ischemia showed that careful predilatation with a cutting balloon followed by a drug-eluting balloon use was sufficient in the majority of cases, with only 14% requiring stent implantation for acute recoil/coronary dissection.[20] Nevertheless, their reported rate of TLF was still high at 14%, 1-year after the procedure. The present findings highlight the complexity of this lesion subset, for which there are limited evidence-based therapies, and emphasize the need for prospective work around alternative management strategies for isolated SB lesions.

## Limitations

There are several limitations to the present study. First, the outcomes that we report are only in patients treated with PCI; our data do not inform on outcomes of lesions treated medically, which is particularly relevant for isolated SB lesions. Second, although the events in our study were independently adjudicated, it is possible that some events were underreported. However, measures to preserve the quality of data reporting, including on-site and remote monitoring, and close communication with the participating sites were in place. Furthermore, the Medina classification was identified based on data submitted in the electronic case report form and not assessed from a core laboratory. Third, some of the CIs in our results are wide because of the relatively small sample size for individual Medina subgroups. Finally, our findings are based on 1-year outcomes, and it is possible that further prognostic differences are observed between Medina subtypes on longer follow-up.

# CONCLUSIONS

Within prospective multicenter data collection with independent event adjudication, we demonstrated significant prognostic differences among 4000 bifurcation PCI procedures according to coronary bifurcation distribution patterns, with Medina 1.1.1 and Medina 0.0.1 associated with worse clinical outcomes at 1 year, including clinically driven TLR and consequently TLF, compared with other Medina subtypes. The present findings should prompt greater caution among operators when managing these higher-risk lesions.

## ARTICLE INFORMATION

Received January 19, 2022; accepted July 15, 2022.

### Affiliations

Keele Cardiovascular Research Group, Centre for Prognosis Research, Institutes of Applied Clinical Science and Primary Care and Health Sciences, Keele University, Newcastle, United Kingdom (M.O.M., M.A.M.); Institute of Health Informatics, University College London, London, United Kingdom (M.O.M.); Department of Interventional Cardiology and Endovascular Therapeutics, Instituto Cardiovascular de Buenos Aires, Buenos Aires, Argentina (P.L.); Department of Cardiology, Hillel Yaffe Medical Center, Hadera, Israel (A.R.); Albert Schweitzer Ziekenhuis, Dordrecht, the Netherlands (R.M.O.); Cardiology Department, Amphia Hospital Breda, Breda, the Netherlands (A.J.I.); Worcestershire Royal Hospital, Worcester, United Kingdom (H.R.); Medical and Clinical Division, Terumo Europe NV, Leuven, Belgium (F.v.L., R.D.); and Division of Cardiology, University Hospitals, Geneva, Switzerland (M.R.).

### Acknowledgments

The authors thank Terumo Europe for the project management of the study.

### Sources of Funding

None.

### Disclosures

R.D. is an employee of Terumo Europe. F.v.L. is a consultant to Terumo Europe. The remaining authors have no disclosures to report.

### Supplemental Material

Data S1
Tables S1–S6
Figure S1

## REFERENCES

1. Burzotta F, Lassen JF, Lefèvre T, Banning AP, Chatzizisis YS, Johnson TW, Ferenc M, Rathore S, Albiero R, Pan M, et al. Percutaneous coronary intervention for bifurcation coronary lesions: the 15(th) consensus document from the European bifurcation Club. *EuroIntervention.* 2021;16:1307–1317. doi: 10.4244/eij-d-20-00169
2. Sawaya FJ, Lefèvre T, Chevalier B, Garot P, Hovasse T, Morice MC, Rab T, Louvard Y. Contemporary approach to coronary bifurcation lesion treatment. *JACC Cardiovasc Interv.* 2016;9:1861–1878. doi: 10.1016/j.jcin.2016.06.056
3. Ludwig J, Mohamed M, Mamas MA. Left main bifurcation lesions: Medina reclassification revisited-as easy as ABC. *Catheter Cardiovasc Interv.* 2021;97:186–187. doi: 10.1002/ccd.29121
4. Medina A, Suárez de Lezo J, Pan M. A new classification of coronary bifurcation lesions. *Rev Esp Cardiol.* 2006;59:183.
5. Riley RF, Henry TD, Mahmud E, Kirtane AJ, Brilakis ES, Goyal A, Grines CL, Lombardi WL, Maran A, Rab T, et al. SCAI position statement on optimal percutaneous coronary interventional therapy for complex coronary artery disease. *Catheter Cardiovasc Interv.* 2020;96:346–362. doi: 10.1002/ccd.28994
6. Mohamed MO, Polad J, Hildick-Smith D, Bizeau O, Baisebenov RK, Roffi M, Íñiguez-Romo A, Chevalier B, von Birgelen C, Roguin A, et al. Impact of coronary lesion complexity in percutaneous coronary intervention: one-year outcomes from the large, multicentre e-Ultimaster registry. *EuroIntervention.* 2020;16:603–612. doi: 10.4244/eij-d-20-00361
7. Chen S-L, Sheiban I, Xu B, Jepson N, Paiboon C, Zhang J-J, Ye F, Sansoto T, Kwan TW, Lee M, et al. Impact of the complexity of bifurcation lesions treated with drug-eluting stents: the DEFINITION study (definitions and impact of complEx biFurcation lesIons on clinical outcomes after percutaNeous coronary IntervenTIOn using drug-eluting steNts). *JACC: Cardiovasc Interv.* 2014;7:1266–1276. doi: 10.1016/j.jcin.2014.04.026
8. Maeng M, Holm NR, Erglis A, Kumsars I, Niemelä M, Kervinen K, Jensen JS, Galløe A, Steigen TK, Wiseth R, et al. Long-term results after simple versus complex stenting of coronary artery bifurcation lesions: Nordic bifurcation study 5-year follow-up results. *J Am Coll Cardiol.* 2013;62:30–34. doi: 10.1016/j.jacc.2013.04.015
9. Di Gioia G, Sonck J, Ferenc M, Chen SL, Colaiori I, Gallinoro E, Mizukami T, Kodeboina M, Nagumo S, Franco D, et al. Clinical outcomes following coronary bifurcation PCI techniques: a systematic review and network meta-analysis comprising 5,711 patients. *JACC Cardiovasc Interv.* 2020;13:1432–1444. doi: 10.1016/j.jcin.2020.03.054
10. Park TK, Park YH, Song YB, Oh JH, Chun WJ, Kang GH, Jang WJ, Hahn JY, Yang JH, Choi SH, et al. Long-term clinical outcomes of true and non-true bifurcation lesions according to Medina classification-results from the COBIS (COronary BIfurcation stent) II registry. *Circ J.* 2015;79:1954–1962. doi: 10.1253/circj.CJ-15-0264

11. Todaro D, Burzotta F, Trani C, Brugaletta S, De Vita M, Talarico GP, Giammarinaro M, Porto I, Leone AM, Niccoli G, et al. Evaluation of a strategy for treating bifurcated lesions by single or double stenting based on the Medina classification. *Rev Esp Cardiol.* 2009;62:606–614. doi: 10.1016/s1885-5857(09)72224-8
12. Tsuchida K, Colombo A, Lefèvre T, Oldroyd KG, Guetta V, Guagliumi G, von Scheidt W, Ruzyllo W, Hamm CW, Bressers M, et al. The clinical outcome of percutaneous treatment of bifurcation lesions in multivessel coronary artery disease with the sirolimus-eluting stent: insights from the arterial revascularization therapies study part II (ARTS II). *Eur Heart J.* 2007;28:433–442. doi: 10.1093/eurheartj/ehl539
13. Vranckx P, Cutlip DE, Mehran R, Kint PP, Silber S, Windecker S, Serruys PW. Myocardial infarction adjudication in contemporary all-comer stent trials: balancing sensitivity and specificity. addendum to the historical MI definitions used in stent studies. *EuroIntervention.* 2010;5:871–874. doi: 10.4244/eijv5i7a146
14. Suleiman S, Coughlan JJ, Touma G, Szirt R. Contemporary management of isolated ostial side branch disease: an evidence-based approach to Medina 001 bifurcations. *Interv Cardiol.* 2021;16:e06–e06. doi: 10.15420/icr.2020.30
15. Shimada Y, Courtney BK, Nakamura M, Hongo Y, Sonoda S, Hassan AH, Yock PG, Honda Y, Fitzgerald PJ. Intravascular ultrasonic analysis of atherosclerotic vessel remodeling and plaque distribution of stenotic left anterior descending coronary arterial bifurcation lesions upstream and downstream of the side branch. *Am J Cardiol.* 2006;98:193–196. doi: 10.1016/j.amjcard.2006.01.073
16. Kim HY, Doh JH, Lim HS, Nam CW, Shin ES, Koo BK, Lee JM, Park TK, Yang JH, Song YB, et al. Identification of coronary artery side branch supplying myocardial mass that may benefit from revascularization. *JACC Cardiovasc Interv.* 2017;10:571–581. doi: 10.1016/j.jcin.2016.11.033
17. Tan KH, Sulke N, Taub N, Sowton E. Percutaneous transluminal coronary angioplasty of aorta ostial, non-aorta ostial, and branch ostial stenoses: acute and long-term outcome. *Eur Heart J.* 1995;16:631–639. doi: 10.1093/oxfordjournals.eurheartj.a060966
18. Mathias DW, Mooney JF, Lange HW, Goldenberg IF, Gobel FL, Mooney MR. Frequency of success and complications of coronary angioplasty of a stenosis at the ostium of a branch vessel. *Am J Cardiol.* 1991;67:491–495. doi: 10.1016/0002-9149(91)90009-a
19. Zimarino M, Corazzini A, Ricci F, Di Nicola M, De Caterina R. Late thrombosis after double versus single drug-eluting stent in the treatment of coronary bifurcations: a meta-analysis of randomized and observational studies. *JACC Cardiovasc Interv.* 2013;6:687–695. doi: 10.1016/j.jcin.2013.03.012
20. Vaquerizo B, Fernández-Nofreiras E, Oategui I, Suarez de Lezo J, Rumoroso JR, Martín P, Routledge H, Tizón-Marcos H. Second-generation drug-eluting balloon for ostial side branch lesions (001-bifurcations): mid-term clinical and angiographic results. *J Interv Cardiol.* 2016;29:285–292. doi: 10.1111/joic.12292

# Supplemental Material

## List of e-Ultimaster Centres and Investigators

**ARGENTINA:** Fundación Favaloro: Oscar Mendiz; Hospital Universitario Austral: Juan Manuel Telayna; Clinica Centro Médico Privado Junin: José Magni; Instituto Cardiovascular de Buenos Aires: Fernando Cura; Sanatorio San Miguel: Juan Lloberas; **ARMENIA:** Astghik Medical Center (Natali Farm): Mikayel Adamyan; Medical Center Gyumri CJSC: Davit Minasyan; Qancor Cardiovascular MC LLC: Shahen Khachatryan; Republican Medical Center Armenia CJSC: Boghos Sarkissian; Yerevan State Medical University Hospital: Hamayak Sisakian; **AUSTRIA**: AKH Linz: Clemens Steinwender; Medical University Vienna (AKH): Irene Lang; Medizinische Universität Graz: Gabor Toth-Mayor; **BANGLADESH:** National Heart Foundation Hospital and Research Institute: Fazila Tun-Nesa Malik; **BELARUS:** City Clinical Emergency Hospital: Alexander Beimanov; RSPC: Oleg Polonetsky; **BELGIUM:** AZ Sint Lucas: Jan Nimmegeers; CHR de La Citadelle: Suzanne Pourbaix; Hôpital Ambroise Paré de Mons: Stéphane Carlier; CHU Charleroi: Adel Aminian; CHU UCL Mont Godinne Namur: Antoine Guédès; Epicura Hornu: Philippe Decroly; Imelda Ziekenhuis: Willem De Wilde; Jan Yperman Ziekenhuis: Dries De Cock; OLVZ Aalst: Bernard De Bruyne; UCL Saint Luc: Joelle Kefer; **BRAZIL:** Eurolatino Natal Pesquisas Medicas (Eurolatino Natal Medical Research): Maria Sanali Paiva; Hospital E Maternidade Dr. Christóvão Da Gama: Bruno Palmieri Bernardi; Hospital Felicio Rocho: Jamil Abdalla Saad; Hospital Moinhos de Vento: Marco Vugman Waistein; Hospital Monte Sinai: Gustavo De Moraes Ramalho; Hospital Santa Cruz: Roberto Otsubo; Hospital São Vicente de Paulo: Rogério Tumelero, Alexandre Tognon; Paraná Medical Research Center: Marcos Franchetti; Unicor: João Eduardo Tinoco De Paula; Unimed Joinville: Bruno Cupertino Migueletto; **BULGARIA:** Mbal Haskovo: Sevdalin Topalov; Mbal Montana City Clinic Sveti Georgi: Krasimir Pandev; Mbal Sveta Karidad, Plovdiv: Dimitar Karageorgiev; Mbal Sveta Petka Vidin: Diana Trendafilova-Lazaroba; Specialized Cardiology Hospital For Active Treatment: Angel Mitov; Trakiya Hospital, Stara Zagora: Borislov Borisov; Umhat Alexandrovska: Dobrin Vassilev; Umhat St.Ekaterina: Julia Jorgova-Makedonska; **CHILE:** Clinica Bicentenario: Carlos Romero; Clinica Santa Maria: Pablo Pedreros; Hospital Clínico San Borja Arriaran: Gabriel Maluenda; Hospital Guillermo Grant Benavente: Luis Perez; Hospital Regional de Antofagasta: Bernhard Westerberg; Hospital Regional Puerto Montt: Victor David Assef; Hospital San Juan de Dios: Angel Puentes; **COLOMBIA:** Centro Cardiovascular de Caldas: Hugo Castaño; Clinica Shaio: Pablo Castro; Fundación Cardiovascular de Colombia (Bucaramanga): Tamara Gorgadze; Instituto del Corazon Bucaramanga: Boris Eduardo Vesga, Hector Hernandez; **CZECH REPUBLIC:** St Anne's University Hospital Brno: Ladislav Groch; Kardiologie na Bulovce: Miroslav Erbrt; Karlovarská Krajská Nemocnice: Alexandr Schee; FNKV Hospital: Viktor KočKa; Krajska Nemocnice T. Bati: Zdenek Coufal; **EGYPT:** Al Hayat Hospital: Hany Ragy; Al Nakheel Hospital: Yasser Sadek; Dr Ahmed Abdel Aziz Multicenter: Mohamed Abdel Aziz; Dr Hussien Heshmat – As Salam International Hospital: Hussien Heshmat; El Marwa Hospital: Mounir Asman; Italian Hospital: Ihab Daoud; L-Fouad Cardiac Center: Ahmed Emara; Dr Hisham Ammar Multicenter: Hisham Ammar; Police Hospital: Mohamed Helal; Dr. Tarek Rasid; Um El Korra M Setiha Hospital: Mohamed Setiha; Nile Badrawy Hospital: Sameh Ahmed Salama; Wadi El Neel: Hazem Khamis; **ESTONIA**: North-Estonia Medical Center: Peep Laanmets; **FRANCE:** Centre D'exploration-Chirurgie Cardio-Vasculaire: Jean-Louis Leymarie; CH Bretagne Atlantique: Emmanuelle Filippi; CH de Marne La Vallée: Simon Elhadad; CH de Montreuil: Chaib Aures; CH Haguenau: Fabien De Poli; Groupe Hospitalier de la Rochelle Ré Aunis: Charlotte Trouillet; CH La Timone Marseille: Jean-Louis Bonnet; CH Louis Pasteur-Le Coudray: Grégoire Rangé; CH de Pau: Nicolas Delarche; CH René Dubos Pontoise: Francois Funck; CH St Joseph St Luc Lyon: Olivier Dubreuil; CH Sud Francilien: Pascal Goube; CH Valence: Stanislas Champin; CH Yves Le Foll - Saint Brieuc: Denis Amer Zabalawi; CHD Vendée La Roche Sur Yon: Emmanuel Boiffard; CH Général de Saint Quentin: Pierre Henon, Florent Chevalier; CHIC Quimper: Thierry Joseph; CHR Orleans Cardiologie: Olivier Bizeau; CHU Angers: Alain Furber; CHU Caen: Farzin Beygui; CHU Clermont-Ferrand: Pascal Motreff; CHU de Poitiers:

Sebastien Levesque; Clinique Ambroise Paré: Julien Rosencher; Clinique Diaconat Fonderie Mulhouse: Pradip Kumar Sewoke; Clinique du Millénaire Montpellier: Christophe Piot; Clinique Du Pont de Chaume Montauban: Laurent Delorme; Clinique Louis Pasteur Essey les Nancy: Max Amor; Clinique Rhône Durance: Gilles Bayet; Clinique Saint-Laurent: Yves Biron; Clinique St Hilaire Rouen: Matthieu Godin; Clinique St Joseph: Julien Jeanneteau; GCS Cardiologique de Bayonne: Jean Luc Banos; Groupe Hopitalier Paris Saint Joseph: Romain Cador; Groupement Mutualiste de Grenoble: Jacques Monsegu; Hopital Privé Claude Galien Quincy: Stéphane Champagne; Hopital Albert Schweitzer GHCA Colmar: Plastaras Philoktimon; Hôpital Europøen de Paris la Roseraie: Hakim Benamer; Hopital Privé Dijon Bourgogne: Philippe Brunel; Hopital Privé Jacques Cartier Massy: Thomas Hovasse; Hopital Privé La Louviere-Lille: Fabrice Leroy; Hopital Privé Saint Martin: Guillaume Lecoq; Hôpital Privé St Martin de Pessac: Levy Raphy; Hôpital Privé St Martin de Pessac: Bernard Karsenty; Institut Arnault Tzanck St Laurent du Var: Alexandre Avran; Le Confluent Nouvelles Cliniques Nantaises: Ashok Tirouvanziam; Nouvel Hopital Civil de Strasbourg: Olivier Morel; Pôle Santé République Clermont Ferrand: Pascal Barraud; Polyclinique Les Fleurs: Philippe Commeau; GEORGIA: Joann Medical Center (JAMC): Lasha Chantladze; **HUNGARY** Pándy Kálmán Hospital: Jambrik Zoltan; Markusovszky University Teaching Hospital: Lajos Nagy; Moritz Kaposi General Hospital: Andras Vorobcsuk; PECS University: Ivan Horvath; Semmelweis University: Bela Merkely; Szabolcs - Szatmar - Bereg County Hospital and University Teaching Hospital: Kôszegi Zsolt; **ICELAND** Landspitali National University Hospital of Iceland: Ingibjörg Jóna Guðmundsdóttir; **INDIA** Dayanand Medical College: Gurpreet Singh Wander; Fortis Hospital: R. Keshava; G. Kuppuswamy Naidu Memorial Hospital: Rajpal Abhaichand; H .J. Doshi Ghatkopar Hindusabha Hospital: Anil Potdar; Heart & General Hospital: Prakash Chandwani; Kamalnayan Bajaj Hospital, Aurangabad: Ajit Bhagwat; Krishna Institute of Medical Sciences: Rajendra Kumar Premchand; Madras Medical Mission: Ajit Mullasari; Maharaja Agrasen Hospital: B B Chanana; Max Super Specialty Hospital: Viveka Kumar; Medanta Hospital: Praveen Chandra; BM Birla Heart Research Cente: Ashwani Mehta; Sree Chitra Tirunal Institute of Medical Sciences & Technology: Bijulal Sasidharan; Wockhardt Hospital: Prashant Jagtap; **INDONESIA** Awal Bros Hospital: Bambang Budiono; Binawaluya Cardiac Center: Muhammad Munawar; RSUPN Dr. Cipto Mangunkusumo Hospital: Muhammad Yamin; Dr. Soetomo General Hospital: Yudi Her Oktaviono; Dr. Wahidin Sudirohusodo General Hospital- Awal Bros Hospital: Abdul Hakim Alkatiri; Medistra Hospital: Teguh Santoso; National Cardiovascular Center Harapan Kita Hospital: Doni Firman; Saiful Anwar General Hospital: Sasmojo Widito; **IRELAND** Cork University Hospital: Eugene McFadden; University Hospital Galway: Jim Crowley; University Hospital Limerick: Thomas Kiernan; **ISRAEL** Assaf Harofeh Medical Center: Minha Saar; Galilee Medical Center: Marc Brezins; Rambam Medical Center: Ariel Roguin; Ziv Medical Center: Majdi Halabi; **JAPAN** Gunma Prefectural Cardiovascular Center: Ren Kawaguchi; Higashi Takarazuka Satoh Hospital: Satoru Otsuji; Iwaki Kyoritsu General Hospital: Yoshito Yamamoto; Kakogawa Central City Hospital: Makoto Kadotani; Kansai Rosai Hospital: Takayuki Ishihara; Kokura Memorial Hospital: Kenji Ando; Komaki City Hospital: Katsuhiro Kawaguchi; Kouseikai Takai Hospital: Yasunori Nishida; Mie Heart Center: Hideo Nishikawa; Mimihara General Hospital: Shozo Ishihara; Okamura Memorial Hospital: Yasuhiro Tarutani; Osaka General Medical Center: Takashi Morita; Osaka Rosai Hospital: Masami Nishino; Saiseikai Senri Hospital: Keiji Hirooka; Saiseikai Yamaguchi General Hospital: Shiro Ono; Saiseikai Yokohama City Eastern Hospital: Yoshiaki Ito; Saitama Cardiovascular And Respiratory Center: Makoto Muto; Sakurabashi Watanabe Hospital: Kenshi Fujii; Sapporo Higashi Tokushukai Hospital: Seiji Yamazaki; Seirei Hamamatsu General Hospital: Hisayuki Okada; Seirei Yokohama Hospital: Kazuhiro Ashida; Shonan Kamakura General Hospital: Shigeru Saito; Showa University Fujigaoka Hospital: Hiroshi Suzuki; Tokai University Hachiouji Hospital: Takashi Matsukage; **JORDAN** Jordan Hospital: Imad Alhaddad; **KAZAKHSTAN** Aktobe Regional Hospital: Aidos Taumov; Cardiology Center Petropavl: Maxat Kudratullayev; City Hospital #2: Marat Alikhanov; Clinical Center of Cardiac Surgery and Transplantation: Vadim Seisembekov; Jsc Nat. Scient. Cardiosurgery Ctr.: Marat Aripov; Medical University Clinic West Kazakhstan: Dauren Teleuov; National Surgery Center Almaty: Bauyrzhan

Ormanov; Pavlodar Regional Cardiologic Center: Ruslan Baisebenov; Regional Cardiosurgery Center: Azamat Kenzhinovich Zhashkeyev; Rudnyi City Hospital: Azamat Yerzhanov; The Almaty City Heart Center: Orazbek Sakhov; Semey State Medical University, Interventional Cardiology Dpt: Ersin Sabitov; **KUWAIT** Sabah Al Ahmad Cardiac Center: Vladimir Kotevski; **LEBANON** Hôpital Abou Jaoudé: Daou Abdo; Labib Medical Center: Ahmad Serhal; **LITHUANIA** Hospital Of Lithuanian University Of Health Sciences Kauno klinikos: Ramunas Unikas; Klaipeda Seamen's Hospital: Aurimas Knokneris; **MACEDONIA** City General Hospital: Vladimir Ristovski; University Clinic Of Cardiology: Sasko Kedev; **MALAYSIA** Desa Park City: Chong Yoon Sin; Hospital Serdang: Abdul Kahar Ghapar; Hospital Sultanah Bahiyah: Abd Syukur Bin Abdullah; Hospital Tengku Ampuan Afzan: Siti Khairani bt Zainal Abidin; HSC Medical Center: Tee Chee Hian; UiTM Sg. Buloh Campus: Nicholas Chua Yul Chye; **MEXICO** Clinica Hospital San Jose de Navojoa: Santiago Sandoval Navarrete; Hospital Fray Juan de San Miguel de Uruapan: Juan Jorge Beltran Ochoa; Hospital Star Medica Merida: Sergio Alonso Villareal Umaña; Casa del Corazon de la Peninsula de Yucatan SCP: Carlos Ramon Rodas Caceres; **MOROCCO** Cherradi_Clinique Agdal: Rhizlan Cherradi; Clinique Achifaa de Casablanca: Anass Assaidi; Clinique Grant Atlas: Dounia Benzaroual; Clinique Internationale de Marrakech: Fahd Chaara; **NETHERLANDS** Albert Schweitzer Ziekenhuis: Martijn Scholte; Amphia Ziekenhuis: Alexander J.J. Ijsselmuiden; Catharina Ziekenhuis: W.A.L. Pim Tonino; Jeroen Bosch Ziekenhuis: Jawed Polad; Jacob van Eck; Maasstad Ziekenhuis: Pieter Cornelis Smits; Meander MC: Fabrizio Spano; Medisch Centrum Haaglanden: Lucas H. Savalle; Medisch Spectrum Twente, Enschede: Clemens Von Birgelen; Rijnstate Ziekenhuis: Peter W. Danse; Scheper Hospital: Gillian Jessurun; Zorgsaam Ziekenhuis Zeeuws-Vlaanderen: Pieter Bisschops; **OMAN** Muscat Private Hospital: Amr Hassan; **POLAND** Insytut Kardiologii im. Prymasa Tys ąclecia Stefana Kardynała Wyszyńskiego: Adam Witkowski; Miedziowe Centrum Zdrowia: Adrian Wlodarczak; Szpital Kliniczny Przemienienia Panskiego Um Im. K. Marcinkowskiego W Poznaniu: Maciej Lesiak; PORTUGAL CHLN Norte Hospital Santa María: Pedro Canas Da Silva; **ROMANIA** Centrele de Excelenta Ares: Alexandru Voican; Clinicile Icco S.R.L.: Mihai Ursu; Cordismed Timisoara: Milovan Slovenski; Spitalul Judetean de Urgenta Sibiu: Ioan Bitea Cornel**; SAUDI ARABIA** Dallah Hospital, Riyadh: Samih Lawand; King Fahad Cardiac Center: Tarek Kashour; Prince Abdullah Bin Abdul Aziz Musad Cardiac Center: Muhammad Aurangzaib Mughal; **SERBIA** Cardiovascular Institute Dedinje: Dragan Sagic; Clinical Center Kragujevac: Nikola Jagic; Cardiology Clinic, Clinical Centre of Serbia: Vladan Vukcevic; Kbc Zvezdara: Alexandar Davidovic; CHC Bezanijska Kosa: Sasa Hinic; **SLOVAKIA** Stredodlovensky Ustav Srdcovych A Cievnych Chorob: Martin Hudec; **SOUTH AFRICA** Ethekwini Hospital & Heart Centre: Shiraz Gafoor; Ismail Soosiwala; Milpark Hospital: Graham Cassel; Netcare Greenacres Hospital: Martin Tawanda Butau; Netcare Union Hospital: Jean-Paul Theron; Netcare Unitas Hospital: Jean Vorster; Netcare Unitas Hospital: Pieter Blomerus; Netcare Unitas Hospital: Iftikar Osman Ebrahim; Netcare Unitas Hospital: Jacobus Badenhorst; **SPAIN** Bellvitge University Hospital: Joan Antonio Gomez; Complexo Hospitalario Universitario A Coruña (CHUAC): Nicolás Vázquez Gonzalez; Hospital 12 Octubre: Fernando Sarnago; Hospital Cabueñes: Iñigo Lozano; Hospital Clínico Lozano Blesa de Zaragoza: José Ramón Ruiz Arroyo; Hospital Clínico Universitario de Santiago de Compostela: Ramiro Trillo Nouche; Clinico Universitario Valencia: Juan Sanchís; Hospital de Cruces-Barakaldo: Juan Alcibar; Hospital Universitario Donostia: Mariano Larman; Hospital de Galdakao: José Ramón Rumoroso; Hospital de La Cruz Roja de Córdoba: José Suárez de Lezo; Hospital de León: Maria López Benito; Hospital de Mérida: Pablo Cerrato Garcia; Hospital de Navarra: Baltasar Lainez; Hospital del Mar: Beatriz Vaquerizo; Hospital Fundacion Alcorcon: Javier Botas; Hospital G. Trias I Pujol: Eduard Fernández Nofrerias; Hospital General Castellón: Pascual Baello Monge; Hospital General Ciudad Real: Fernando Lozano Ruiz-Poveda; Hospital General de Albacete: Jesus Maria Jimenez Mazuecos; Hospital General Universitario de Burgos: Javier Robles; Hospital Infanta Cristina: José Ramon Lopez Minguez; Hospital Juan Ramón Jiménez: Pepi Garcia; Clinica La Luz: Jorge Palazuelos; Hospital Manises: Gema Miñana; Hospital Marqués de Valdecilla: Jose Javier Zueco; Hospital Meixoeiro-Medtec: Andrés Iñiguez Romo; Hospital Moncloa: Eulogio Garcia Fernandez; Hospital Puerta de Hierro: Javier Goicolea; Hospital Reina Sofia

de Córdoba: Manuel Pan; Clínica San Fransisco de Asis: Arturo García Touchard; Hospital San Pedro: Javier Fernández; Hospital San Pedro de Alcantara-Caceres: Javier Fernandez Portales; Hospital San Rafael: Gonzalo Peña; Hospital Sant Pau: Antonio Peñaranda Serra; Hospital Santa Lucía de Cartagena Hospital Nostra Señora Rossell: José Domingo Cascón; Hospital Txagorritxu: Alfonso Torres; Hospital Universitario de Gran Canaria Dr Negrin: Pedro Martin Lorenzo; Hospital Universitario de Guadalajara: Javier Balaguer Requena; Hospital Universitario Lucus Augusti (HULA): Raymundo Ocaranza Sanchez; Hospital Universitario Miguel Servet (H.U.M.S.): Jose Antonio Diarte de Miguel; Hospital Vall d'Hebron: Bruno García Del Blanco; Hospital Virgen Arrixaca: Eduardo Pinar; Hospital Virgen de La Salud: P. José Moreu Burgos; Instituto Cardiologico Hospital Campo Grande: Juan Manuel Duran; San Juan de Alicante: Ramón López Palop; Universitario Central de Asturias: César Moris-De La Tassa; SWEDEN Gävle Sjukhus: Robert Kastberg; Mälarsjukshuet: Finn Hjortevang; Skaraborgs Sjukhus v Skövde: Jason Stewart; Sundvalls Sjukhus: Espen Haugen; Universitets Sjukhuset I Örebro: Ole Fröbert; Västmanlads Sjukhus Västerås: Amra Kåregren; **SWITZERLAND** Cardiocentro Lugano, Ticino: Giovanni Pedrazzini; Herz Gefäss Zentrum Zürich: Peter Wenaweser; Hôpital de La Tour: Edoardo De Benedetti; Hôpitaux Universitaires de Genève: Maro Roffi; Kantonsspital Baselland: Gregor Leibundgut; Kantonsspital Frauenfeld Spital Thurgau AG: Michael Neuhaus; Kantonsspital Luzern: Florim Cuculi; THAILAND Central Chest Institute Of Thailand: Wirash Kehasukcharoen; HRH Princess Maha Chakri Sirindhorn Medical Center (Nakornayok): Arthit Wongsoasup; Paolo Memorial Hospital Phaholyothin: Niphonth Srisuwanunt; **TUNISIA** Dr. Mohamed Drissa Clinique Hannibal Lac 2: Mohamed Akram Drissa; Dr. Ben Chedli Tarek - Soukra Medical: Ben Chedli Tarek; Dr. Bouziri - Clinique Générale Et Cardiovasculaire de Tunis: Sami Bouziri; Dr. Elyes Kharrat - Bassatine Clinic: Elyes Kharrat; Polyclinique El bassatine_Dr. Mohamed Najeh Abid: Mohamed Najeh Abid; Clinique Générale et Cardiovasculaire de Tunis _Dr. Saloua Trabelsi: Saloua Trabelsi; Polyclinique El Bassatine: Rridha Ennouri; **UKRAINE** Heart Institute: Andriy Khohlov; NAMS Amosov | Emergency Endovascular Surgery Department: Sergii Salo; NAMS Amosov | X-Ray Diagnostics And Invasive Cardiology Department: Yevhenii Aksonov; S.P.M.C. of Pediatric Cardiology and Cardiac Surgery: Georgiy Mankovskiy; **UNITED ARAB EMIRATES** Al Noor Hospital - Airport: Mohammad Andron; Al Qassimi Hospital: Arif Al Nooryani; Al Zahra Private Hospital, Dubai: Syed Nazir; Belhoul Speciality Hospital, Dubai: Muhammad Adnan Raufi; Dr. Sulaiman Al Habib: Albert Alahmar; Dubai Hospital: Hesham Ahmed Osman; Iranian Hospital, Dubai: Seyed Bagher Tabatabaei; Lifecare Hospital: Khaled Galal; Prime Hospital, Dubai: Murali Krishna; Rashid Hospital: Fahad Omar Baslaib; UNITED KINGDOM Essex Cardiothoracic Centre, Basildon: Rohan Jagathesan; Bedford Hospital: Ramesh de Silva; Blackpool Victoria Hospital: Jonas Eichhofer; Bradford Teaching Hospitals: John Kurian; Croydon University Hospital: Sanjay Kumar; Dorset County Hospital: Javed Iqbal; Eastbourne District General Hospital: David Walker; Freeman Hospital: Rajiv Das; GBS Re Bucks Healthcare NHS Trust (Buckinghamshire, Wycombe): Piers Clifford; James Cook University Hospital: David Austin; Kettering General Hospital: Javed Ehtisham; Kings Mill Hospital: Ifti Fazal; Lincoln County Hospital: Kelvin Lee; Lister Hospital, Stevenage: Paul Kotwinski; The Royal Wolverhampton Hospitals: Shahzad Munir; Norfolk And Norwich University Hospital: Alisdair Ryding; Northwick Park Hospital: Ahmed Elghamaz; Plymouth Hospital: Girish Viswanathan; Queen Elizabeth Hospital, Birmingham: Sagar Doshi; Queens Medical Center Nottingham: Sachin Jadhav; Royal Berkshire Hospital: Nicos Spyrou; Royal Blackburn Hospital: John Mcdonald; Royal Bournemouth And Christchurch Hospitals NHS Foundation Trust: Suneel Talwar; Royal Brompton And Harefield: Robert Smith; Royal Cornwall Hospitals: Sen Devadathan; Derby Teaching Hospitals: Kamal Chitkara; The Royal Free Hospital: Sundeep Kalra; Royal Gwent Hospital, Newport: James Cullen; Royal Stoke University Hospital: Mamas Mamas; Royal Sussex Hospital, Brighton: David Hildick Smith; Royal United Hospital, Bath: Kevin Carson; Salisbury District Hospital: Tim Wells; Sandwell And West Birmingham Hospitals: Chetan Varma; Sheffield Teaching Hospital: James Richardson; Tunbridge Wells Hospital: Clive Lawson; UH Coventry and Warwickshire: Rajathurai Thirumaran; University Hospital South Manchester: Hussain Contractor; University Hospital Of Wales: Rito Mitra; University Hospitals Of Leicester: Ian Hudson; West Middlesex Hospital: Sukhinder Nijjer;

Western Sussex Hospitals - Worthing Hospital: Nicholas Pegge; Worcestershire Acute Hospitals NHS Trust: Helen Routledge; Wrightington Hospital: V J Karthikeyan; **UZBEKISTAN** Republic Specialized Center of Surgery: Mirjamol Mirumarovich Zufarov; **VIETNAM** Thong Nhat Hospital: Nguyen Van Tan

**Table S1. Type of 2-stent techniques used for patients with a true bifurcation lesion.**

| **True** | Missing | Crush | Culotte | Kissing stents | Other | T-stenting | TAP | V-stenting |
|---|---|---|---|---|---|---|---|---|
| 0.1.1 | 1.5% (2/133) | 13.5% (18/133) | 12.8% (17/133) | 9.8% (13/133) | 6.0% (8/133) | 21.1% (28/133) | 15.0% (20/133) | 20.3% (27/133) |
| 1.0.1 | 0.9% (1/116) | 12.9% (15/116) | 6.0% (7/116) | 6.9% (8/116) | 40.0% (46/116) | 24.1% (28/116) | 5.2% (6/116) | 4.3% (5/116) |
| 1.1.1 | 1.0% (5/479) | 19.0% (91/479) | 13.8% (66/479) | 7.9% (38/479) | 10.4% (50/479) | 24.4% (117/479) | 19.0% (91/479) | 4.4% (21/479) |
| **All** | 1.1% (8/728) | 17.0% (124/728) | 12.4% (90/728) | 8.1% (59/728) | 14.3% (104/728) | 23.8% (173/728) | 16.1% (117/728) | 7.3% (53/728) |

**Table S2. Type of 2-stent techniques used for patients with a non-true bifurcation lesion.**

| **Non-true** | Missing | Crush | Culotte | Kissing stents | Other | T-stenting | TAP | V-stenting |
|---|---|---|---|---|---|---|---|---|
| 0.0.1 | 0.0% (0/16) | 6.3% (1/16) | 18.8% (3/16) | 6.3% (1/16) | 18.8% (3/16) | 37.5% (6/16) | 6.3% (1/16) | 6.3% (1/16) |
| 0.1.0 | 0.0% (0/10) | 0.0% (0/10) | 40.0% (4/10) | 0.0% (0/10) | 30.0% (3/10) | 10.0% (1/10) | 10.0% (1/10) | 10.0% (1/10) |
| 1.0.0 | 0.0% (0/12) | 8.3% (1/12) | 0.0% (0/12) | 25.0% (3/12) | 0.0% (0/12) | 58.3% 7/12) | 0.0% (0/12) | 8.3% (1/12) |
| 1.1.0 | 5.0% (3/60) | 5.0% (3/60) | 8.3% (5/60) | 6.7% (4/60) | 38.3% 23/60) | 20.0% 12/60) | 11.7% (7/60) | 5.0% (3/60) |
| **All** | 3.1% (3/98) | 5.1% (5/98) | 12.2% (12/98) | 8.2% (8/98) | 29.6% (29/98) | 26.5% (26/98) | 9.2% (9/98) | 6.2% (6/98) |

**Table S3. Clinical outcomes at 1-year for true and non-true bifurcations according to stent strategy (1 vs. 2 stents) and Medina classification subtype.**

| | True bifurcation | | | | | | Non-true bifurcation | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | **0.1.1** | **0.1.1** | **1.0.1** | **1.0.1** | **1.1.1** | **1.1.1** | **0.0.1** | **0.0.1** | **0.1.0** | **0.1.0** | **1.0.0** | **1.0.0** | **1.1.0** | **1.1.0** |
| | 1-stent n=141 | 2-stent n=129 | 1-stent n=211 | 2-stent n=108 | 1-stent n=879 | 2-stent n=463 | 1-stent n=42 | 2-stent n=16 | 1-stent n=367 | 2-stent n=10 | 1-stent n=302 | 2-stent n=12 | 1-stent n=962 | 2-stent n=60 |
| TLF, % [n] | 2.8 [4] | 2.3 [3] | 4.3 [9] | 5.6 [6] | 5.5 [48] | 6.7 [31] | 11.9 [5] | 12.5 [2] | 3.5 [13] | 0.0 [0] | 2.3 [7] | 8.3 [1] | 4.9 [47] | 3.3 [2] |
| POCE, % [n] | 6.4 [9] | 5.4 [7] | 9.0 [19] | 12.0 [13] | 9.8 [86] | 12.3 [57] | 14.3 [6] | 12.5 [2] | 7.4 [27] | 0.0 [0] | 6.3 [19] | 8.3 [1] | 10.0 [96] | 3.3 [2] |
| Cardiac death, % [n] | 0.7 [1] | 0.0 [0] | 0.5 [1] | 2.8 [3] | 2.3 [20] | 2.4 [11] | 4.8 [2] | 6.3 [1] | 2.2 [8] | 0.0 [0] | 1.0 [3] | 0.0 [0] | 1.6 [15] | 0.0 [0] |
| TV-MI, % [n] | 0.0 [0] | 0.0 [0] | 1.4 [3] | 1.9 [2] | 1.6 [14] | 1.9 [9] | 2.4 [1] | 0.0 [0] | 0.3 [1] | 0.0 [0] | 1.0 [3] | 0.0 [0] | 1.4 [13] | 1.7 [1] |
| CD-TLR, % [n] | 2.1 [3] | 2.3 [3] | 3.8 [8] | 1.9 [2] | 3.1 [27] | 3.7 [17] | 7.1 [3] | 6.3 [1] | 1.1 [4] | 0.0 [0] | 1.0 [3] | 8.3 [1]* | 2.9 [28] | 1.7 [1] |
| ST [def/prob], % [n] | 0.0 [0] | 0.0 [0] | 0.5 [1] | 2.8 [3] | 0.8 [7] | 1.3 [6] | 0.0 [0] | 0.0 [0] | 0.3 [1] | 0.0 [0] | 1.0 [3] | 0.0 [0] | 0.7 [7] | 0.0 [0] |

*p-value for 1 vs. 2-stent strategy: <0.05, non-significant for all other outcomes.

**Table S4. Clinical outcomes at 1-year per type of 2-stent technique.**

| **2-stents** | Crush | Culotte | Kissing stents | Other | T-stenting | TAP | V-stenting | p-value |
|---|---|---|---|---|---|---|---|---|
| TLF | 8.6% (10/117) | 9.2% (8/87) | 1.7% (1/58) | 4.0% (4/101) | 5.4% (9/167) | 5.3% (6/114) | 2.1% (1/48) | 0.29 |
| POCE | 11.1% (13/117) | 20.7% (18/87) | 8.6% (5/58) | 10.9% (11/101) | 7.8% (13/167) | 10.5% (12/114) | 6.3% (3/48) | 0.07 |
| All death | 2.6% (3/117) | 9.2% (8/87) | 3.5% (2/58) | 7.9% (8/101) | 1.2% (2/167) | 0.9% (1/114) | 0.0% (0/48) | <0.01 |
| Cardiac death | 2.6% (3/117) | 4.6% (4/87) | 1.7% (1/58) | 4.0% (4/101) | 0.6% (1/167) | 0.9% (1/114) | 0.0% (0/48) | 0.21 |
| All MI | 3.4% (4/117) | 3.5% (3/87) | 0.0% (0/58) | 1.0% (1/101) | 2.4% (4/167) | 0.9% (1/114) | 2.1% (1/48) | 0.59 |
| CD-TLR | 6.0% (7/117) | 3.5% (3/87) | 0.0% (0/58) | 0.0% (0/101) | 3.6% (6/167) | 4.4% (5/114) | 2.1% (1/48) | 0.17 |
| Stent thrombosis (def/prob) | 1.7% (2/117) | 2.3% (2/87) | 0.0% (0/58) | 1.0% (1/101) | 1.2% (2/167) | 1.8% (2/114) | 0.0% (0/48) | 0.87 |

**Table S5. Clinical outcomes at 1-year for bifurcation lesions involving the left main per Medina classifications.**

| **Left main** | 0.0.1 | 0.1.0 | 0.1.1 | 1.0.0 | 1.0.1 | 1.1.0 | 1.1.1 | p-value |
|---|---|---|---|---|---|---|---|---|
| TLF | 0.0% (0/3) | 9.4% (3/32) | 4.2% (1/24) | 2.8% (1/36) | 5.3% (1/19) | 7.1% (11/154) | 13.5% (24/178) | 0.25 |
| POCE | 0.0% (0/3) | 18.8% (6/32) | 4.2% (1/24) | 2.8% (1/36) | 15.8% (3/19) | 13.6% (21/154) | 21.9% (39/178) | 0.04 |
| All death | 0.0% (0/3) | 9.4% (3/32) | 4.2% (1/24) | 0.0% (0/36) | 15.8% (3/19) | 4.6% (7/154) | 9.6% (17/178) | 0.17 |
| Cardiac death | 0.0% (0/3) | 6.3% (2/32) | 4.2% (1/24) | 0.0% (0/36) | 5.3% (1/19) | 3.3% (5/154) | 6.2% (11/178) | 0.70 |
| All MI | 0.0% (0/3) | 3.1% (1/32) | 0.0% (0/24) | 0.0% (0/36) | 0.0% (0/19) | 0.7% (1/154) | 2.8% (5/178) | 0.62 |
| CD-TLR | 0.0% (0/3) | 0.0% (0/32) | 0.0% (0/24) | 2.8% (1/36) | 0.0% (0/19) | 3.9% (6/154) | 7.3% (13/178) | 0.29 |
| Stent thrombosis (def/prob) | 0.0% (0/3) | 0.0% (0/32) | 0.0% (0/24) | 0.0% (0/36) | 0.0% (0/19) | 0.7% (1/154) | 1.1% (2/178) | 0.97 |

**Table S6. Clinical outcomes at 1-year for bifurcation lesions involving the LAD per Medina classifications.**

| **LAD** | 0.0.1 | 0.1.0 | 0.1.1 | 1.0.0 | 1.0.1 | 1.1.0 | 1.1.1 | p-value |
|---|---|---|---|---|---|---|---|---|
| TLF | 7.7% (5/65) | 2.8% (7/252) | 2.6% (5/193) | 3.5% (6/171) | 6.0% (11/183) | 3.3% (20/599) | 5.5% (47/858) | 0.11 |
| POCE | 9.2% (6/65) | 5.2% (13/252) | 5.7% (11/193) | 8.2% (14/171) | 12.6% (23/183) | 7.5% (45/599) | 8.9% (76/858) | 0.11 |
| All death | 3.1% (2/65) | 2.4% (6/252) | 0.5% (1/193) | 2.9% (5/171) | 1.6% (3/183) | 1.2% (7/599) | 3.2% (27/858) | 0.12 |
| Cardiac death | 3.1% (2/65) | 1.6% (4/252) | 0.0% (0/193) | 1.8% (3/171) | 0.0% (0/183) | 0.8% (5/599) | 2.1% (18/858) | 0.08 |
| All MI | 0.0% (0/65) | 0.8% (2/252) | 0.0% (0/193) | 1.2% (2/171) | 3.8% (7/183) | 1.8% (11/599) | 2.2% (19/858) | 0.07 |
| CD-TLR | 4.6% (3/65) | 1.2% (3/252) | 2.6% (5/193) | 1.8% (3/171) | 5.5% (10/183) | 2.3% (14/599) | 2.8% (24/858) | 0.15 |
| Stent thrombosis (def/prob) | 0.0% (0/65) | 0.4% (1/252) | 0.0% (0/193) | 1.2% (2/171) | 1.6% (3/183) | 0.7% (4/599) | 1.1% (9/858) | 0.52 |

**Figure S1. Bifurcation subtypes as per the Medina classification.**