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ORIGINAL RESEARCH

Abbreviated or standard antiplatelet therapy after PCI in HBR patients with chronic kidney disease: a prespecified analysis from the MASTER DAPT trial

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ABSTRACT

BACKGROUND: Abbreviated antiplatelet therapy (APT) can reduce bleeding without increasing ischaemic harm in high bleeding risk (HBR) patients undergoing percutaneous coronary intervention (PCI). The impact of chronic kidney disease (CKD) on the safety and effectiveness of abbreviated APT remains unknown.

AIMS: We aimed to investigate the comparative effectiveness of abbreviated (1 month) versus standard (≥3 months) APT in HBR patients with and without CKD.

METHODS: This was a prespecified analysis from the MASTER DAPT trial, which randomised 4,579 HBR patients (1,428 [31%] with CKD) to abbreviated or standard APT. CKD was defined as an estimated glomerular filtration rate <60 mL/min/1.73 m^2. Co-primary outcomes were net adverse clinical events (NACE; a composite of all-cause death, myocardial infarction [MI], stroke, and major bleeding), major adverse cardiac or cerebral events (MACCE; all-cause death, MI and stroke), and Bleeding Academic Research Consortium (BARC) 2, 3, or 5 bleeding at 11 months.

RESULTS: NACE did not significantly differ with abbreviated and standard APT among CKD patients (hazard ratio [HR] 0.91, 95% confidence interval [CI]: 0.66-1.24) and non-CKD patients (HR 0.96, 95% CI: 0.73-1.27; $p_{interaction}$=0.78). Similarly, MACCE did not differ in CKD patients (HR 0.91, 95% CI: 0.64-1.27) and non-CKD patients (HR 1.09, 95% CI: 0.78-1.51; $p_{interaction}$=0.45). Abbreviated APT was associated with consistently lower BARC 2, 3, or 5 bleeding in both patients with CKD (HR 0.74, 95% CI: 0.52-1.07) and without it (HR 0.66, 95% CI: 0.51-0.85; $p_{interaction}$=0.59).

CONCLUSIONS: Abbreviated APT was associated with similar NACE and MACCE rates and reduced bleeding compared with standard APT in HBR patients undergoing PCI, regardless of the presence or absence of CKD. (ClinicalTrials.gov: NCT03023020)

KEYWORDS: chronic kidney disease; dual antiplatelet therapy; high bleeding risk; percutaneous coronary intervention

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Chronic kidney disease (CKD) is a highly prevalent clinical condition, affecting more than 850 million individuals worldwide with a global median prevalence of 9.5%[1,2]. The burden of renal dysfunction is even more prevalent among patients with coronary artery disease (CAD) or acute coronary syndrome (ACS) undergoing percutaneous coronary intervention (PCI)[3]. Accumulating evidence suggests that CKD patients undergoing PCI face increased risks of both ischaemic and bleeding complications[4,5]. This "double hazard" may be explained by altered platelet function and coagulation pathways[6], reduced responsiveness to antiplatelet agents[7], and a greater burden of comorbidities in this population[5]. Furthermore, CKD is associated with more advanced and complex CAD, including a higher prevalence of multivessel disease, left main involvement, chronic total occlusions, extensive calcifications, and increased plaque burden[8,9].

Patients undergoing PCI typically require a combination of aspirin and a $P2Y_{12}$ inhibitor, commonly referred to as dual antiplatelet therapy (DAPT), to mitigate the risk of ischaemic recurrences, including stent thrombosis (ST)[10,11]. However, this ischaemic protection offered by DAPT must be balanced against an elevated risk of bleeding, especially in patients at high bleeding risk (HBR)[12]. As such, the optimal DAPT duration after PCI for the prevention of ischaemic and bleeding complications in HBR-CKD patients remains a clinical conundrum.

The Management of High Bleeding Risk Patients Post Bioresorbable Polymer Coated Stent Implantation With an Abbreviated Versus Prolonged DAPT Regimen (MASTER DAPT) study demonstrated that abbreviated (1-month) DAPT was non-inferior to treatment continuation for at least 2 additional months for net and major adverse clinical events and was associated with reduced major or clinically relevant non-major bleeding (CRNMB) in HBR patients treated with a biodegradable-polymer sirolimus-eluting stent[13-15]. In this prespecified analysis of MASTER DAPT, we sought to investigate whether the treatment effects of abbreviated versus standard antiplatelet therapy (APT) differ according to the presence of CKD in this HBR population.

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Impact on daily practice

Patients with chronic kidney disease (CKD) undergoing percutaneous coronary intervention (PCI) have higher risks of ischaemic and bleeding events than non-CKD subjects. The optimal duration of dual antiplatelet therapy (DAPT) in CKD patients with concomitant high bleeding risk (HBR) features represents a clinical conundrum. In this prespecified analysis from the MASTER DAPT trial, an abbreviated DAPT duration was associated with comparable major and net adverse clinical events and consistently reduced bleeding, compared with treatment continuation, for at least two additional months, regardless of renal function. These findings support an abbreviated DAPT regimen in HBR-CKD patients after PCI with biodegradable-polymer sirolimus-eluting stents. Future studies should investigate the safety and efficacy of abbreviated DAPT in HBR patients with end-stage renal disease or those on dialysis treatment.

Methods

STUDY DESIGN

This is a prespecified analysis of MASTER DAPT (ClinicalTrials.gov: NCT03023020), an investigator-initiated, randomised, open-label, non-inferiority trial with sequential superiority testing, enrolling an unselected patient population at HBR following implantation of a biodegradable-polymer-coated Ultimaster sirolimus-eluting stent (Terumo). Study methodology and the primary results of MASTER DAPT have been previously reported[13-17]. The trial was approved by the institutional review board at each participating site, and all patients gave written informed consent. An independent data safety monitoring board regularly reviewed the conduct of the trial and patient safety. Study organisation and participating sites are reported in **Supplementary Appendix 1**, along with a complete list of the MASTER DAPT investigators.

STUDY POPULATION

Patients at HBR were deemed eligible for trial participation if they underwent treatment of all coronary lesions requiring revascularisation with an Ultimaster stent for acute or chronic coronary syndromes and remained event-free (including a new ACS, symptomatic restenosis, ST, stroke, or any revascularisation resulting in the prolonged use of DAPT) during the first month after index PCI. Patients were considered at HBR if at least one of the following criteria applied: oral anticoagulant (OAC) therapy for at least 12 months, recent (<12 months) non-access site bleeding episode(s) that required medical attention, previous bleeding episode(s) that required hospitalisation if the underlying cause had not been definitively treated, age ≥75 years, systemic conditions associated with an increased bleeding risk (e.g., haematological or coagulation disorders), documented anaemia, need for chronic treatment with steroids or non-steroidal anti-inflammatory drugs, malignancy (other than skin), stroke at any time or transient ischaemic attack (TIA) in the previous 6 months, or PREdicting bleeding

Abbreviations

APT	antiplatelet therapy	**MACCE**	major adverse cardiac or cerebral events
BARC	Bleeding Academic Research Consortium	**NACE**	net adverse clinical events
CKD	chronic kidney disease	**PCI**	percutaneous coronary intervention
CRNMB	clinically relevant non-major bleeding	**SAPT**	single antiplatelet therapy
DAPT	dual antiplatelet therapy		

Complications In patients undergoing Stent implantation and subsequent DAPT (PRECISE-DAPT) score ≥25[18].

Key exclusion criteria were the implantation of any stent other than the Ultimaster stent within the previous 6 months, a bioresorbable scaffold at any time before the index procedure, or stenting for in-stent restenosis or ST. Detailed inclusion and exclusion criteria are presented in **Supplementary Appendix 1**.

RENAL FUNCTION ASSESSMENT

Renal function was evaluated using the most recent value of serum creatinine after the index PCI prior to hospital discharge. The estimated glomerular filtration rate (eGFR) was calculated using the Modification of Diet in Renal Disease (MDRD) equation. Chronic kidney disease was defined as an eGFR <60 mL/min/1.73 m^2. For subgroup analysis, CKD severity was further stratified into mild-to-moderate CKD (eGFR ≥30 but <60 mL/min/1.73 m^2) and severe CKD (<30 mL/min/1.73 m^2).

RANDOMISATION AND FOLLOW-UP

Patients were centrally randomised (1:1 ratio) to an open-label abbreviated or standard APT regimen 30 to 44 days after the index procedure. Randomisation was concealed using a web-based system; randomisation sequences were computer-generated, blocked (with randomly selected block sizes of 2, 4, or 6), and were stratified by site, history of acute myocardial infarction (MI) within the past 12 months, and clinical indication for at least 12-month OAC.

Patients randomly allocated to the abbreviated treatment group immediately discontinued DAPT and continued single antiplatelet therapy (SAPT) until study completion, except for those receiving OAC, who continued SAPT up to 6 months after the index procedure. Patients allocated to the standard treatment group continued DAPT for at least 5 additional months (i.e., 6 months after the index procedure) or, for those receiving OAC, for at least 2 additional months (i.e., 3 months after the index procedure) followed by SAPT. Follow-up visits took place at 60 days, 150 days, and 335 days (all with a ±14-day window) after randomisation.

OUTCOMES

The three ranked co-primary outcomes were net adverse clinical events (NACE; the composite of death from any cause, MI, stroke, or major bleeding), major adverse cardiac or cerebral events (MACCE; the composite of death from any cause, MI, or stroke), and major or CRNMB (the composite of Bleeding Academic Research Consortium [BARC] Type 2, 3, or 5 bleeding) at 11 months after randomisation (12-month follow-up).

Secondary outcomes included the individual components of the three co-primary outcomes, cardiovascular or non-cardiovascular death, cerebrovascular accidents (CVA; the composite of stroke and TIA), definite or probable ST, and all BARC bleeding events. All events were adjudicated by an independent adjudication committee that was unaware of the treatment allocations. All data were stored at a central database (Department of Clinical Research, University of Bern, Bern, Switzerland).

STATISTICAL ANALYSIS

The data were analysed according to the intention-to-treat principle. Outcomes were assessed separately for patients with or without CKD, by calculating hazard ratios (HRs) with 95% confidence intervals (CIs). For patients with a primary outcome, the time to event was calculated as the difference between the date of occurrence of the outcome event and the date of randomisation, plus 1 day. For patients with incomplete clinical follow-up, the time to censoring was defined as the difference between the dates of the last known clinical status and randomisation, plus 1 day. The Com-Nougue method was used to analyse the time to event, with day 0 defined as the date of randomisation at the 1-month visit and the analysis extending up to 335 days thereafter. Kaplan-Meier calculations included all (first) adjudicated outcome events that occurred between randomisation and 335 days thereafter according to the randomised treatment assignment, irrespective of the DAPT regimen received at the time of the outcome event. HRs and 95% CIs were generated for primary and secondary outcomes with the use of Cox proportional hazards regression analysis, with censoring at the end of the study and at the time of death. P-values for testing the homogeneity of the HR in subgroups of patients (including those with or without a concomitant indication for OAC and by CKD severity) were derived from Cox proportional hazards models, with the interaction term for treatment group (abbreviated vs standard) and CKD (yes vs no) tested using one degree of freedom. The continuous relation between eGFR used as continuous variable with NACE, MACCE, and major or CRNMB was assessed using spline functions. The analyses were carried out using Stata/SE 16.0 (StataCorp).

Results

Among the 4,579 patients enrolled in the MASTER DAPT trial, 1,428 patients (31%) had CKD. Randomisation occurred at a median of 34 days post-PCI (interquartile range: 32 to 39 days) to either an abbreviated (n=2,295 [CKD: n=711; non-CKD: n=1,584]) or a standard (n=2,284 [CKD: n=717; non-CKD: n=1,567]) APT. The composition of DAPT and type of SAPT did not differ between patients with and without CKD **(Supplementary Table 1)**.

BASELINE AND PROCEDURAL CHARACTERISTICS

Patients with CKD were older, more often female, and had a higher cardiovascular risk burden, including arterial hypertension, diabetes mellitus, prior MI or coronary revascularisation, compared with non-CKD patients **(Supplementary Table 2)**. CKD patients had a lower left ventricular ejection fraction (51.2% vs 54.1%; p<0.001), more frequently had haematological or coagulation disorders (20.2% vs 9.2%; p<0.001), and had higher PRECISE DAPT scores (34.24±10.13 vs 23.37±9.59; p<0.001) than non-CKD patients **(Supplementary Table 2)**. The mean creatinine clearance was 47.2±16.1 mL/min/1.73 m^2 and 81.6±18.8 mL/min/1.73 m^2 in CKD and non-CKD patients (p<0.001), respectively. Non-ST-segment elevation ACS was more prevalent in CKD patients than in non-CKD patients (30.5% vs 22.8%; p<0.001), while stable angina was less common (36.3% vs 42.2%; p<0.001).

Supplementary Table 3 shows angiographic and procedural characteristics in CKD and non-CKD patients. CKD patients underwent PCI more frequently via transfemoral access and had a higher prevalence of treated vessels or complex lesions (type B2/C, left main, or lesion requiring rotational atherectomy) than non-CKD patients. Baseline and procedural characteristics according to CKD and the randomly allocated APT were well balanced between the groups except for a lower prevalence of prior CVA, radial access, and multivessel PCI in non-CKD patients treated with abbreviated compared with standard APT **(Supplementary Table 4, Supplementary Table 5)**.

CLINICAL OUTCOMES BY CKD

At 11-month follow-up **(Table 1)**, the incidence of NACE was higher in CKD patients than in non-CKD patients (11.10% vs 6.25%; HR 1.82, 95% CI: 1.48-2.24; p<0.001). The rate of MACCE was also higher in CKD patients compared with non-CKD patients (9.34% vs 4.56%; HR 2.10, 95% CI: 1.66-2.66; p<0.001), whereas major or CRNMB occurred in 119 of 1,428 CKD patients (8.48%) and in 240 of 3,151 non-CKD patients (7.70%; HR 1.10, 95% CI: 0.89-1.38; p=0.375). The rates of death (either cardiovascular or non-cardiovascular), CVA, and MI were higher in CKD patients than in those without CKD. BARC Type 3 or 5 bleeding occurred in 50 of 1,428 CKD patients (3.58%) and in 72 of 3,151 non-CKD patients (2.31%; HR 1.55, 95% CI: 1.08-2.23; p=0.017).

CLINICAL OUTCOMES BY CKD AND RANDOMLY ALLOCATED ANTIPLATELET REGIMENS

Clinical outcomes at 11 months in CKD and non-CKD patients stratified by APT are shown in **Figure 1** and **Figure 2**. NACE did not differ between abbreviated and standard APT among CKD patients (75 [10.60%] vs 83 [11.59%]; HR 0.91, 95%

Table 1. Clinical outcomes at 11 months after randomisation in HBR patients with versus without CKD.

	CKD (n=1,428)	Non-CKD (n=3,151)	Hazard ratio (95% CI)	*p*-value
NACE	158 (11.10)	196 (6.25)	1.82 (1.48-2.24)	<0.001
MACCE	133 (9.34)	143 (4.56)	2.10 (1.66-2.66)	<0.001
Major or CRNMB	119 (8.48)	240 (7.70)	1.10 (0.89-1.38)	0.375
Death	85 (5.97)	71 (2.27)	2.68 (1.96-3.68)	<0.001
Cardiovascular death	43 (3.06)	38 (1.22)	2.54 (1.64-3.92)	<0.001
Non-cardiovascular death	29 (2.09)	28 (0.90)	2.32 (1.38-3.91)	0.001
Undetermined death	13 (0.93)	5 (0.16)	5.83 (2.08-16.34)	0.001
Cerebrovascular accident	23 (1.65)	26 (0.84)	1.99 (1.13-3.48)	0.017
Stroke*	16 (1.15)	19 (0.61)	1.89 (0.97-3.67)	0.061
Ischaemic stroke	13 (0.93)	16 (0.52)	1.82 (0.87-3.78)	0.109
Haemorrhagic stroke	4 (0.29)	2 (0.06)	4.51 (0.83-24.62)	0.082
TIA	7 (0.51)	7 (0.23)	2.25 (0.79-6.40)	0.130
Myocardial infarction	49 (3.52)	60 (1.93)	1.84 (1.26-2.68)	0.002
Definite or probable ST	9 (0.65)	14 (0.45)	1.44 (0.62-3.32)	0.397
Definite ST	4 (0.29)	14 (0.45)	0.64 (0.21-1.94)	0.428
Probable ST	5 (0.36)	0 (0)	24.27 (1.34-438.62)	0.003
Bleeding (BARC classification)				
BARC Type 1	51 (3.64)	123 (3.95)	0.92 (0.67-1.28)	0.627
BARC Type 2	74 (5.29)	180 (5.78)	0.91 (0.70-1.20)	0.517
BARC Type 3	44 (3.15)	68 (2.19)	1.45 (0.99-2.11)	0.057
BARC Type 3a	26 (1.86)	30 (0.96)	1.94 (1.15-3.28)	0.014
BARC Type 3b	14 (1.01)	27 (0.87)	1.16 (0.61-2.21)	0.656
BARC Type 3c	5 (0.36)	11 (0.35)	1.02 (0.35-2.92)	0.978
BARC Type 4	0 (0)	0 (0)		
BARC Type 5	6 (0.44)	4 (0.13)	3.37 (0.95-11.95)	0.060
BARC Type 5a	1 (0.07)	1 (0.03)	2.24 (0.14-35.80)	0.569
BARC Type 5b	5 (0.37)	3 (0.10)	3.75 (0.90-15.70)	0.070
BARC Type 3 or 5	50 (3.58)	72 (2.31)	1.55 (1.08-2.23)	0.017

Data are given as n (%). No. of first events of each type (Kaplan-Meier failure %). Hazard ratio (95% CI) from Cox time-to-first event analyses in the ITT population. Continuity corrected risk ratios (95% CI) in case of zero events with Fisher's exact test p-value. Interaction p-value testing for the modifying effect of CKD (yes or no) on the hazard ratio scale. *Includes undetermined strokes. Event counts for stroke subtypes reflect all the subtypes separately (e.g., no hierarchical counting was applied). BARC: Bleeding Academic Research Consortium; CI: confidence interval; CKD: chronic kidney disease; CRNMB: clinically relevant non-major bleeding; HBR: high bleeding risk; HR: hazard ratio; ITT: intention-to-treat; MACCE: major adverse cardiac and cerebral events; NACE: net adverse clinical events; ST: stent thrombosis; TIA: transient ischaemic attack

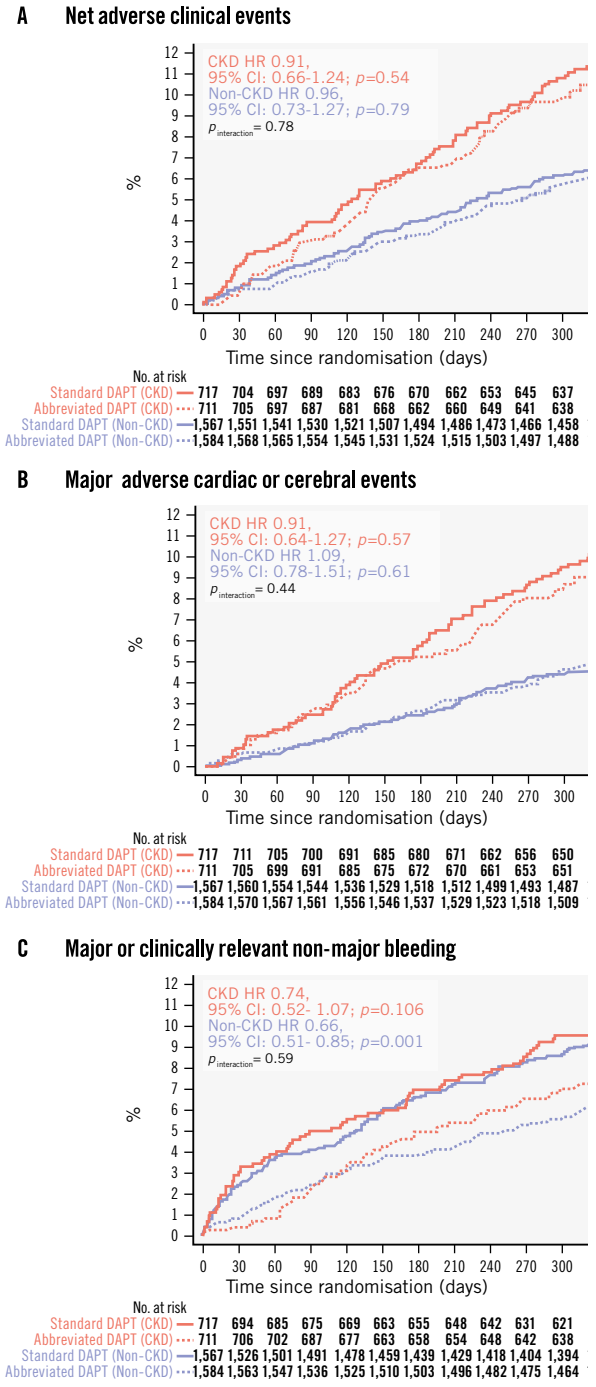


Figure 1. *Kaplan-Meier curves showing the three co-primary outcomes. Kaplan-Meier curves for NACE (A), MACCE (B) and major or clinically relevant non-major bleeding (C). CI: confidence interval; CKD: chronic kidney disease; DAPT: dual antiplatelet therapy; HR: hazard ratio; MACCE: major adverse cardiac or cerebral events; NACE: net adverse clinical events*

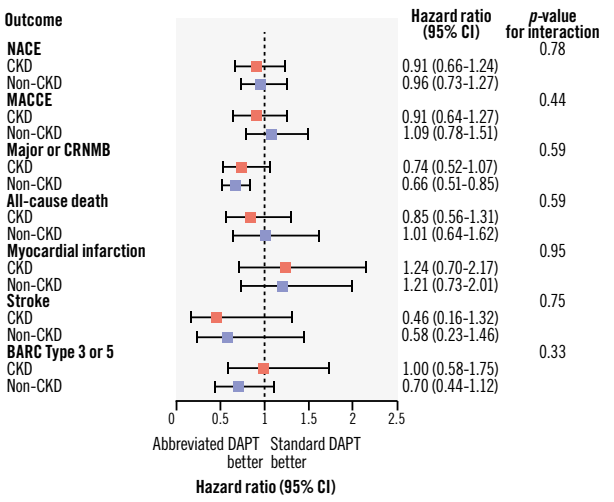


Figure 2. *Main outcomes of abbreviated versus standard antiplatelet therapy in CKD and non-CKD patients. Abbreviated and standard DAPT were compared based on CKD status, with hazard ratios and 95% confidence intervals (CIs) for the three co-primary outcomes and their components (all-cause death, myocardial infarction, stroke, Bleeding Academic Research Consortium [BARC] Type 3 or 5 bleeding). CKD: chronic kidney disease; CRNMB: clinically relevant non-major bleeding; DAPT: dual antiplatelet therapy; MACCE: major adverse cardiac and cerebral events; NACE: net adverse clinical events*

CI: 0.66-1.24; p=0.540) and non-CKD patients (97 [6.15%] vs 99 [6.35%]; HR 0.96, 95% CI: 0.73-1.27; p=0.792), with no significant heterogeneity of the treatment effect at interaction testing ($p_{interaction}$=0.780) **(Supplementary Table 6)**. Similarly, MACCE did not differ with abbreviated and standard APT among CKD (63 [8.90%] vs 70 [9.78%]; HR 0.91, 95% CI: 0.64-1.27; p=0.570) and non-CKD patients (75 [4.76%] vs 68 [4.37%]; HR 1.09, 95% CI: 0.78-1.51; p=0.610), with no heterogeneity ($p_{interaction}$=0.445) **(Supplementary Table 6)**. Major or CRNMB was consistently reduced with abbreviated versus standard APT in CKD patients (51 [7.33%] vs 68 [9.63%]; HR 0.74, 95% CI: 0.52-1.07; p=0.106) and non-CKD patients (97 [6.19%] vs 143 [9.23%]; HR 0.66, 95% CI: 0.51-0.85; p=0.001; $p_{interaction}$=0.590) **(Supplementary Table 6)**. There was no evidence of heterogeneity of the treatment effects (all $p_{interaction}$>0.1) by CKD for any of the individual components of the co-primary endpoints or other secondary endpoints **(Supplementary Table 6, Supplementary Figure 1)**. Major or CRNMB reduction with abbreviated DAPT was mainly driven by lower rates of BARC Type 2 bleeding, both in CKD (HR 0.68, 95% CI: 0.43-1.08; p=0.100; risk difference –1.95) and non-CKD patients (HR 0.65, 95% CI: 0.48-0.87; p=0.004; risk difference –2.38; $p_{interaction}$=0.880) **(Supplementary Table 6)**.

OUTCOMES IN CKD AND NON-CKD PATIENTS WITH OR WITHOUT A CLINICAL INDICATION FOR OAC

Among patients with an indication for OAC **(Supplementary Table 7)**, NACE, MACCE and major or CRNMB were similar between abbreviated and standard APT regardless of the presence or absence of CKD.

Among patients without an indication for OAC **(Supplementary Table 8)**, major or CRNMB was significantly

reduced with abbreviated APT in both CKD (HR 0.61, 95% CI: 0.36-1.04; p=0.072) and non-CKD patients (HR 0.52, 95% CI: 0.36-0.76; p=0.001), with no interaction (p=0.653).

ADDITIONAL POST HOC ANALYSES

Of the 1,428 CKD patients, 1,245 (87.2%) had mild-to-moderate CKD (eGFR 30-59 mL/min/1.73 m^2), while 183 (12.8%) had severe CKD (eGFR <30 mL/min/1.73 m^2). Event rates increased progressively with worsening renal function **(Figure 3, Supplementary Table 9)**. No significant heterogeneity of the treatment effect between CKD severity and the randomly allocated APT regimen for any of the three co-primary endpoints was observed ($p_{interaction}$>0.1) **(Figure 3, Supplementary Table 9)**. However, the absolute risk difference in major or CRNMB associated with abbreviated APT was greater in patients with severe CKD (–8.05%) compared with mild-to-moderate CKD (–1.5%) and non-CKD (–3.04%) **(Supplementary Table 9)**.

Figure 4 shows spline functions for NACE, MACCE, and major or CRNMB using eGFR as a continuous variable. The risks of NACE and MACCE did not significantly differ with abbreviated versus standard APT as eGFR decreased. However, the absolute risk reduction in major or CRNMB with abbreviated APT progressively increased as a function of renal impairment severity **(Figure 4)**.

Discussion

To the best of our knowledge, this is one of the largest analyses investigating the comparative effectiveness and safety of abbreviated versus standard APT in HBR patients with or without CKD undergoing PCI **(Central illustration)**. The main findings from this prespecified analysis can be summarised as follows:

1. Patients with CKD experienced a 2-fold higher risk of net and major adverse cardiac or cerebral events at 1 year post-PCI, due to higher rates of death, MI, CVA, and major bleeding compared with patients without CKD.
2. There was no evidence of heterogeneity between CKD status and the effect of randomly allocated APT regimens on the three co-primary outcomes, suggesting that abbreviated APT was consistently associated with similar NACE and MACCE rates and lower major or CRNMB rates compared with standard APT, in both CKD and non-CKD patients.
3. Stepwise increases in both bleeding and ischaemic risks were observed with worsening renal function. The absolute and relative benefits of abbreviated APT were more pronounced in patients with severe CKD compared with mild-moderate dysfunction and non-CKD patients.

The observation of similar NACE and MACCE rates and a consistent reduction in bleeding events with abbreviated compared with standard APT, irrespective of CKD status, challenges the notion that patients with CKD require prolonged DAPT for ischaemic protection. Current guidelines provide no specific recommendations for DAPT duration after PCI based on renal function[19,20]. Although CKD was not an inclusion criterion of the MASTER DAPT trial, renal dysfunction is considered a major (if severe) or minor (if moderate) criterion for HBR by the Academic Research Consortium[21], and eGFR is one of the five items included in the PRECISE DAPT score[18]. Likewise, CKD is listed among the thrombotic risk enhancers according to the European Society of Cardiology guidelines, which recommend that a second antithrombotic agent in addition to aspirin should or may be considered in patients with and without complex CAD with a Class IIa and IIb recommendation, respectively[19,20]. However, these recommendations apply to non-HBR patients.

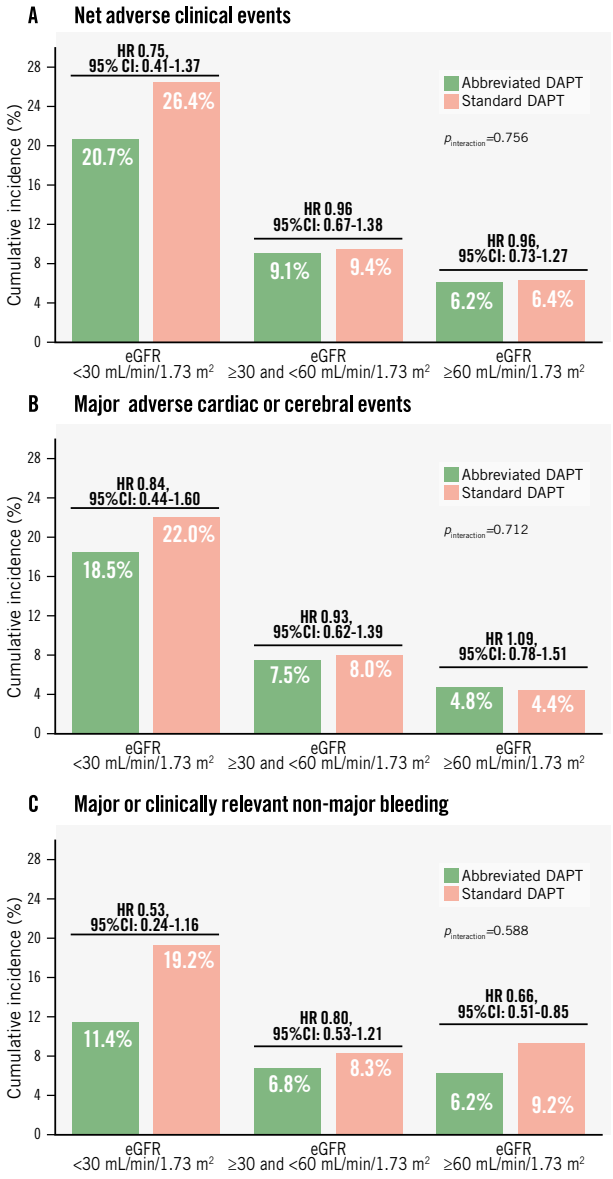


Figure 3. *Interaction between CKD severity and antiplatelet therapy on the three co-primary efficacy outcomes. The x-axis shows the categories of the patients according to CKD severity, and the y-axis shows the event rates of the co-primary efficacy outcomes: net adverse clinical events (A), major adverse cardiac or cerebral events (B), and major or clinically relevant non-major bleeding (C). CI: confidence interval; CKD: chronic kidney disease; DAPT: dual antiplatelet therapy; eGFR: estimated glomerular filtration rate; HR: hazard ratio*

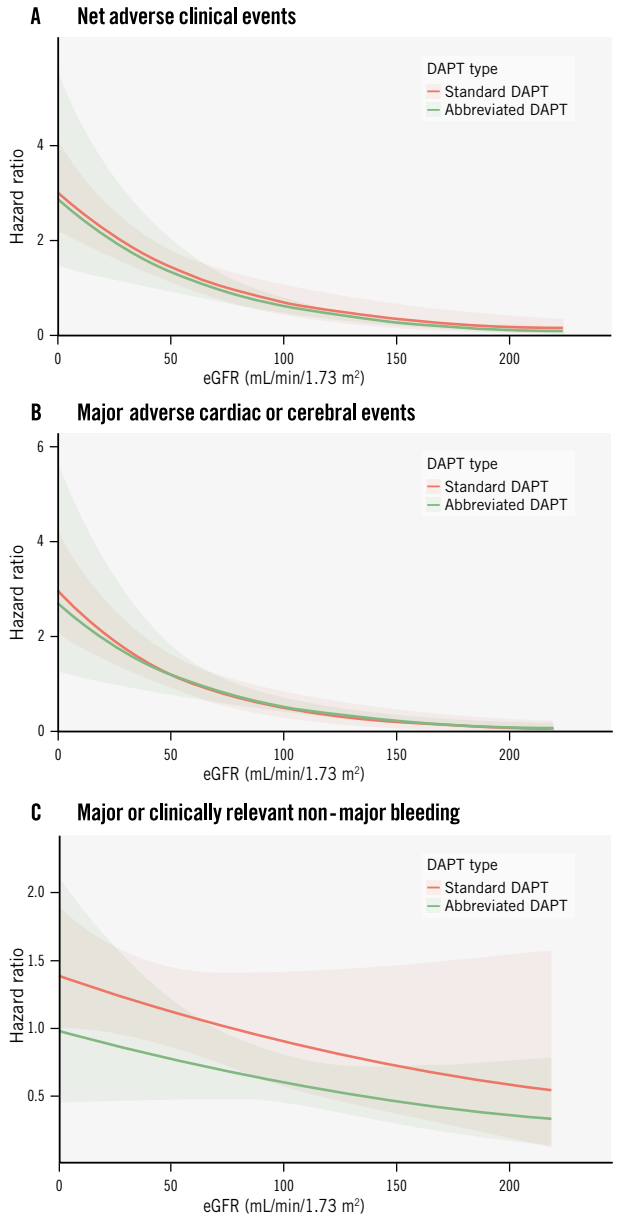


Figure 4. *Spline functions of the three co-primary outcomes. Spline functions of net adverse clinical events (A), major adverse cardiac or cerebral events (B), and major or clinically relevant non-major bleeding (C) in patients randomly allocated to abbreviated or standard DAPT according to eGFR. DAPT: dual antiplatelet therapy; eGFR: estimated glomerular filtration rate*

At secondary endpoint analyses, we observed greater benefits in terms of absolute and relative risk reduction with abbreviated APT in patients with severely impaired renal function. Patients with severe CKD had significantly higher risks of adverse events compared with patients with moderate CKD or preserved renal function. In this high-risk subgroup, abbreviated APT was associated with a greater bleeding benefit without an incremental ischaemic risk compared with standard APT. Although the upper bound of the 95% CI slightly exceeded unity, likely due to the limited sample size, the results remain reassuring.

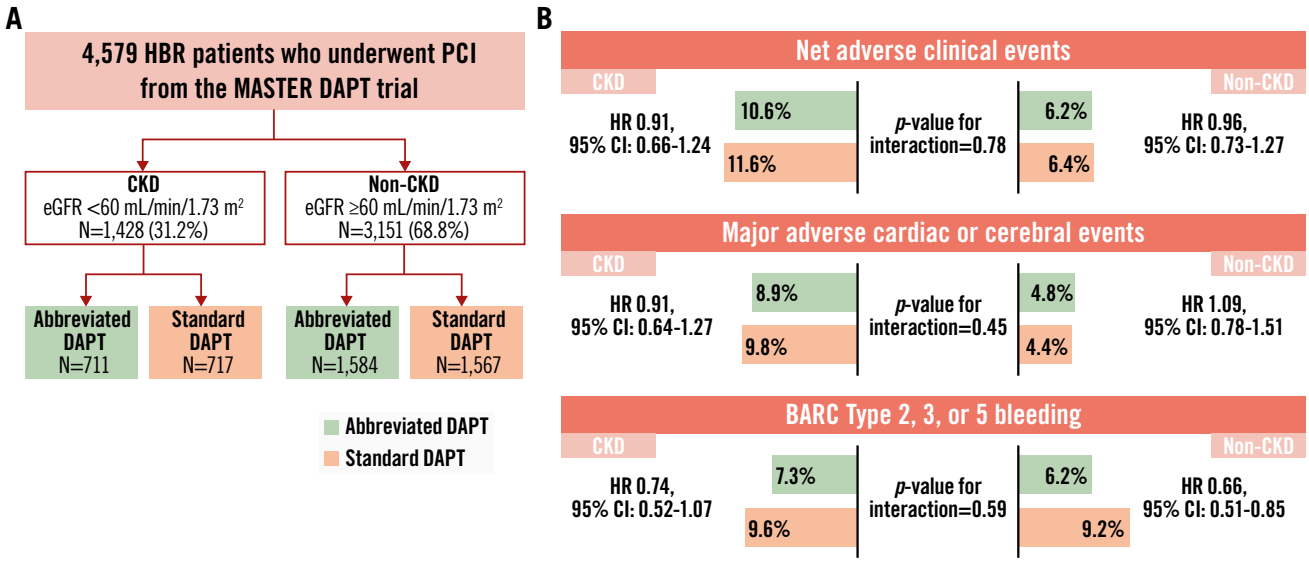
Our findings align with those from the XIENCE Short DAPT clinical programme encompassing data from three prospective single-arm studies, which included 3,286 HBR PCI patients (43.6% with CKD) treated with 1- or 3-month DAPT followed by aspirin monotherapy[22]. One- versus 3-month DAPT was associated with comparable rates of the composite primary endpoint of all-cause death or MI in CKD (adjusted HR 0.86, 95% CI: 0.60-1.22) and non-CKD patients (adjusted HR 1.15, 95% CI: 0.77-1.73; $p_{interaction}$=0.299). BARC Type 2 to 5 bleeding was consistently reduced with 1- compared with 3-month DAPT in CKD (HR 0.74, 95 CI: 0.52-1.04) and non-CKD patients (HR 0.70, 95% CI: 0.48-1.03), with no significant heterogeneity at interaction testing ($p_{interaction}$=0.462). Our results expand on these findings in a larger HBR cohort predominantly managed with $P2Y_{12}$ inhibitor monotherapy (clopidogrel in 53.9%, ticagrelor in 13.6%, prasugrel in 1.2%) rather than aspirin (28.8%).

Conversely, the Swedish Web-System for Enhancement and Development of Evidence-Based Care in Heart Disease Evaluated According to Recommended Therapies (SWEDEHEART) registry showed that prolonged (>3 months) compared with 3-month DAPT was associated with lower risks of the composite endpoint of death, MI, or ischaemic stroke (adjusted HR 0.84, 95% CI: 0.73-0.96) and net adverse events (HR 0.84, 95% CI: 0.78-0.91), with an enhanced magnitude of the treatment effects as renal function worsens[23]. However, prolonged DAPT was associated with a 31% relative increase in bleeding compared with 3-month DAPT[23]. The SWEDEHEART study was not randomised and exclusively included ACS patients without HBR features. The proportion of ACS patients was 50.8% in MASTER DAPT and 35.8% in the XIENCE Short DAPT clinical programme. In a *post hoc* analysis of the Harmonizing Optimal Strategy for Treatment of Coronary Artery Diseases Trial -Comparison of REDUCTION of PrasugrEl Dose and POLYmer TECHnology in ACS Patients (HOST REDUCE POLYTECH RCT) Trial, which randomised ACS patients to DAPT de-escalation (prasugrel 5 mg from 1 month onwards) versus conventional therapy with aspirin and prasugrel 10 mg for 12 months[24], prasugrel de-escalation was associated with a greater bleeding benefit and similar ischaemic risk as renal function worsened[24]. These results suggest that patients with severe CKD may derive enhanced bleeding benefit without ischaemic harm from DAPT de-escalation.

In MASTER DAPT, patients underwent PCI with biodegradable-polymer sirolimus-eluting stents, which limits the generalisability of these findings to other stent platforms. However, while the majority of randomised trials provided evidence on stent type selection by comparing different stent platforms in HBR patients[25-27], MASTER DAPT is the only trial investigating the optimal duration of DAPT in patients who were selected specifically for HBR.

Although there are no dedicated trials for CKD patients, subgroup analyses of randomised clinical trials have also investigated the efficacy and safety of $P2Y_{12}$ inhibitor monotherapy after 1 to 3 months of DAPT in CKD and non-CKD patients. In the GLOBAL LEADERS trial, which randomised 15,991 patients with chronic or ACS to 1-month

Abbreviated or standard antiplatelet therapy in high bleeding risk patients with and without CKD.



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A) Study design. (B) Study outcomes. BARC: Bleeding Academic Research Consortium; CI: confidence interval; CKD: chronic kidney disease; DAPT: dual antiplatelet therapy; eGFR: estimated glomerular filtration rate; HBR: high bleeding risk; HR: hazard ratio; PCI: percutaneous coronary intervention

DAPT (aspirin and ticagrelor) followed by 23-month ticagrelor monotherapy versus 12-month DAPT followed by aspirin alone[28], no significant differences in the primary endpoint of all-cause mortality or new Q-wave MI nor BARC Type 3 or 5 bleeding were observed[28]. A prespecified subgroup analysis showed no heterogeneity of the treatment effects between the randomly allocated APT and CKD status[29]. Ticagrelor-based monotherapy was associated with a significantly lower risk of BARC Type 3 or 5 bleeding as eGFR decreased[29]. However, these findings should be interpreted with caution due to the overall negative trial outcome and the low prevalence of CKD (14% of the overall study population). Our results further extend these findings to a contemporary HBR population. In the Ticagrelor With Aspirin or Alone in High-Risk Patients after Coronary Intervention (TWILIGHT) trial, ticagrelor monotherapy after 3 months of DAPT was associated with a lower risk of BARC Type 2 to 5 bleeding and a similar risk of ischaemic events in CKD and non-CKD patients compared with ticagrelor plus aspirin[30]. In an individual patient data meta-analysis comprising 25,960 patients from 6 trials, ticagrelor monotherapy was non-inferior to 12-month DAPT for the composite primary endpoint of death, MI, or stroke (HR 0.89, 95% CI: 0.74-1.06; p for non-inferiority=0.004) and superior for major bleeding (HR 0.47, 95% CI: 0.36-0.62; p<0.001)[31]. Compared with 12-month DAPT, clopidogrel monotherapy was associated with lower bleeding (HR 0.49, 95% CI: 0.30-0.81; p=0.006) but failed to show non-inferiority for the composite primary ischaemic endpoint (HR 1.37, 95% CI: 1.01-1.87; p for non-inferiority>0.99). Subgroup analyses for the primary endpoint demonstrated consistent treatment effects in patients with and without CKD without significant heterogeneity at interaction testing ($p_{interaction}$=0.74 for ticagrelor; $p_{interaction}$=0.51 for clopidogrel).

Taken together, this body of evidence – including our current analysis – suggests that CKD, while a marker of elevated ischaemic risk, should not be the sole determinant for prolonging DAPT in HBR patients. Abbreviated APT strategies are supported and may be particularly favourable in patients with impaired renal function.

Limitations

This study has some limitations. First, the MASTER DAPT trial was not powered for non-inferiority comparisons of NACE and MACCE with abbreviated versus standard APT in the CKD subgroup, and randomisation was not stratified by CKD. Thus, our findings should be considered exploratory and hypothesis-generating. Dedicated prospective studies are required to assess the risks and benefits of an abbreviated versus standard APT regimen in this patient population. Second, the trial included only patients who were free of ischaemic and bleeding events in the first month post-PCI using biodegradable-polymer sirolimus-eluting stents. Therefore, these findings may not apply to patients experiencing early adverse events, those at low bleeding risk, or those treated with other stent platforms. Third, in this analysis, CKD was based on a single post-PCI creatinine measurement without accounting for baseline values. Finally, patients with in-stent

restenosis or ST were excluded, limiting generalisability to these populations.

Conclusions

In this prespecified analysis of the MASTER DAPT trial, abbreviated APT was associated with similar NACE and MACCE and reduced major or CRNMB compared with standard APT, regardless of renal function. In HBR patients with severe CKD undergoing biodegradable-polymer sirolimus-eluting coronary stent implantation, abbreviated APT appears particularly beneficial, offering significant bleeding reduction without increased ischaemic harm. These findings support the consideration of a shortened APT regimen in HBR-CKD patients after PCI with biodegradable-polymer sirolimus-eluting stents.

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Conflict of interest statement

A. Landi declares that he has received consulting fees from Terumo, outside the submitted work. F. Mahfoud has been supported by Deutsche Forschungsgemeinschaft (SFB TRR219, Project-ID 322900939), and Deutsche Herzstiftung; Saarland University has received scientific support from Ablative Solutions, Medtronic, and Recor Medical; up to May 2024, he received speaker honoraria/consulting fees from Ablative Solutions, AstraZeneca, Inari, Medtronic, Merck & Co, Novartis, Philips, and Recor Medical. D. Heg and K. Chalkou are employed by the Department of Clinical Research (DCR), University of Bern, which has a staff policy of not accepting honoraria or consultancy fees; however, the DCR is involved in the design, conduct, and analysis of clinical studies funded by not-for-profit and for-profit organisations. In particular, pharmaceutical and medical device companies provide direct funding to some of these studies. For an up-to-date list of our conflicts of interest see https://dcr.unibe.ch/services/declaration_of_interest/index_eng.html. E. Barbato declares grants or contracts from Pfizer; honoraria from Abbott, Boston Scientific, and Insight Lifetech; and participation on advisory boards for Novo Nordisk and Abbott, outside the submitted work. N. Kukreja has received grants from DalCor Pharma, the Population Health Research Institute, the European Cardiovascular Research Institute, and Daiichi Sankyo; and has received personal fees from AstraZeneca and Pfizer, outside the submitted work. A. Tirouvanziam reports receiving consultant and proctor (TAVI) fees from Edwards Lifesciences, outside the submitted work. P.C. Smits reports personal consulting or speaker fees from Terumo, Abiomed, and OpSense; grants and personal consulting fees from Abbott, MicroPort, and Daiichi Sankyo; and grants from SMT, outside the submitted work. M. Valgimigli reports grants and/or personal fees from AstraZeneca, Terumo, Alvimedica/CID, Abbott, Daiichi Sankyo, Bayer, CoreFlow, Idorsia Pharmaceuticals, Universität Basel Department Klinische Forschung, Vifor, Bristol-Myers Squibb SA, Biotronik, Boston Scientific, Medtronic, Vesalio, Novartis, Chiesi, and PhaseBio, outside the submitted work. The other authors have no conflicts of interest relevant to the contents of this paper to declare.

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Supplementary data

Supplementary Appendix 1. MASTER DAPT trial: committees and investigators, additional information on the methods.

Supplementary Table 1. Details on DAPT and SAPT at 1-, 3-, 6- and 12-month study visits in CKD versus non-CKD patients stratified by the randomly allocated antiplatelet regimen.

Supplementary Table 2. Baseline characteristics among CKD and non-CKD patients.

Supplementary Table 3. Procedural characteristics among CKD and non-CKD patients.

Supplementary Table 4. Baseline characteristics by randomised antiplatelet regimen and CKD status.

Supplementary Table 5. Procedural characteristics by randomised antiplatelet regimen and CKD status.

Supplementary Table 6. Clinical outcomes at 11 months after randomisation in CKD and non-CKD patients randomised to abbreviated versus standard DAPT.

Supplementary Table 7. Clinical outcomes at 11 months after randomisation (12-month follow-up) in CKD and non-CKD patients with a clinical indication for 12-month OAC.

Supplementary Table 8. Clinical outcomes at 11 months after randomisation (12-month follow-up) in CKD and non-CKD patients without a clinical indication for 12-month OAC.

Supplementary Table 9. Clinical endpoints at 11 months after randomisation (12-month follow-up) according to renal dysfunction severity (eGFR thresholds at 45 and 60 mL/min/1.73 m^2).

Supplementary Figure 1. Kaplan-Meier curves for all-cause mortality, myocardial infarction, stroke, and BARC Type 3 or 5 bleeding.

The supplementary data are published online at: https://eurointervention.pcronline.com/doi/10.4244/EIJ-D-25-00566

Supplementary data

Supplementary Appendix 1. MASTER DAPT trial: committees and investigators, additional information on the methods.

Supplementary Appendix 1. MASTER DAPT trial: committees and investigators, additional information on the methods.

MASTER DAPT trial: committees and investigators

Executive committee

Coprincipal Investigator	M. Valgimigli
Coprincipal Investigator	P.C. Smits
Sponsor Representative	G.A. Van Es until June 1, 2018
Sponsor Representative	From June 1 2018, G.B.W.E. Vos
Sponsor Representative	From October 16, 2020, E. Spitzer
Principal Investigators	P. Vrancks; B. Chevalier; Y. Ozaki
Clinical Research Organization Representative	M.C. Morice
Cardiologists	Y. Onuma; E. Frigoli
Bern University Hospital Representative	A. Frenk
Biostatisticians	P. Jüni; J. Tijssen
Terumo Representative (nonvoting member)	D. Paunovic

Steering committee

National Lead Investigators

Bangladesh, India	M.S. Ajit
Kingdom of Bahrain, Saudi Arabia	M. Alasnag
Belgium	J. Bartunek
France	B. Chevalier
Italy	A. Colombo
United Kingdom	D. Hildick-Smith
Portugal, Spain	A. Iñiguez
Austria, Denmark, Estonia, Germany, Sweden	F. Mahfoud
Israel	R. Kornowski
Bulgaria, Czech Republic, Hungary, Poland	M. Lesiak
Singapore, South Korea, Vietnam	P.J. Ong
Japan	Y. Ozaki
Argentina	A.E. Rodriguez
Switzerland	M. Roffi
Australia	C. Schultz
Macedonia, Serbia	G. Stankovic
The Netherlands	P. Tonino

Top enrollers

Inselspital, Bern, Switzerland	Aris Moschovitis
North Estonia Medical Center, Tallinn, Estonia	Peep Laanmets
Interventional Cardiology Unit, Policlinico Casilino, Rome, Italy	Michael Donahue

Data monitoring committee

Chair	M. Bertrand
Biostatistician	S. Pocock
Cardiologist	P. Urban

Clinical events committee

Chair	S. Leonardi
Cardiologists	C. Hanet; R. Lopes; E.P. McFadden; P. Radke; E. Spitzer
Neurologist	R. O. Roine

Safety reporting group (Cardialysis, Rotterdam, The Netherlands)

Boudijn Ladan (Safety Specialist), Laura van der Waal (Safety Assistant), Yvonne Engelbrecht (Safety Assistant), Fred Paddenburg (Safety Manager), Ben Ren, M.D., Ph.D. (Medical Reviewer).

CEC management group (Cardialysis, Rotterdam, The Netherlands)

Ingrid de Zwart (Data Manager), Liliane Elshout (Data Manager), Judith Jonk (Data Manager), Tessa Rademaker-Havinga (Statistician).

Project management

Ria van Vliet (Project Manager, ECRI, Rotterdam, The Netherlands). Marie-Claude Morice (Medical Director, CERC, France), Phani Krishna Kondamudi (Clinical Project Leader, CERC, France), Laure Morsiani (Clinical Operations Manager, CERC, France), Ute Windhövel (Regulatory Affairs Manager, CERC, France). Anita van der Wal (Project Manager, Cardialysis, Rotterdam, the Netherlands), Chantal Bakker (Project Manager, Cardialysis, Rotterdam, The Netherlands). Kazuhiro Minagawa (Project Manager, CVQuest, Tokyo, Japan).

Countries, investigators, and numbers of patients enrolled

Country	Site name	Principal investigator	Patients randomised (n=4579)	Patients consented but not randomised (n=625)
Argentina			16	1
	Buenos Aires, Otamendi Hospital	Dr Juan Mieres	8	
	Buenos Aires, InstitutIo Cardiovascular de Buenos Aires	Dr Fernando Cura	5	1
	Buenos Aires, Clinica IMA	Dr Carlos Fernandez-Pereira	3	
Australia			142	19
	Perth, Royal Perth Hospital-Cardiology Research	Prof. Carl Schultz	66	7
	Wollongong, Wollongong Hospital	Dr Astin Lee	55	6
	Sydney, Prince of Wales Hospital	Dr Nigel Jepson	8	2
	Fitzroy, St Vincent Hospital	Prof. Robert Whitbourn	7	
	Chermside, The Prince Charles Hospital	Dr Owen Christopher Raffel	6	4
Austria			44	11
	Vienna, Wilhelminenspital	Prof. Kurt Huber	29	9
	Vienna, Rudolfstiftung Hospital	Prof. Franz Weidinger	15	2
Bangladesh	Dhaka, National Heart Foundation Hospital & Research Institute	Prof. Fazila-Tun-Nesa Malik	39	1
Belgium			302	51
	Hasselt, Jessa Ziekenhuis	Prof. Pascal Vranckx	91	14
	Bonheiden, Imelda Ziekenhuis	Dr Willem Dewilde	90	14
	Charleroi, CHU de Charleroi – Hopital Civil Marie Curie	Dr Adel Aminian	48	3
	Aalst, OLV Ziekenhuis	Prof. Emanuele Barbato----from 6th Sep 2018 Dr Jozef Bartunek	47	7
	Liege, CHR La Citadelle	Dr Suzanne Pourbaix	24	11
	Brussels, CHU St. Pierre UMC St. Pieter	Dr Panagiotis Xaplanteris	2	2
Bulgaria			183	11

Country	Site name	Principal investigator	Patients randomised (n=4579)	Patients consented but not randomised (n=625)
	Sofia, UMHAT St. Anna	Dr Vasil Velchev	91	
	Plovdiv, MHAT "Sveta Karidad" Plovdiv	Dr Dimitar Karageorgiev	60	10
	Sofia, National Heart Hospital	Dr Hristo Mateev	23	1
	Sofia, Tokuda Hospital	Prof. Valeri Gelev	9	
Czech Republic			134	17
	Brno, University Hospital Brno	Prof. Petr Kala	120	17
	Praha, Na Homolce Hospital	Dr Martin Mates	14	
Denmark	Roskilde, Roskilde Hospital Kogevej	Dr Henning Kelbæk	13	
Estonia	Tallinn, North-Estonia Medical Centre Foundation	Dr Peep Laanmets	259	12
France			578	67
	Massy, Hopital Prive Jacques Cartier	Dr Thomas Hovasse	129	19
	Montauban, Clinique du Pont de Chaume	Dr Laurent Delorme	124	7
	Marseille, CHU La Timone	Prof. Thomas Cuisset	41	
	Annecy, Centre Hospitalier Annecy Genvois	Dr Loïc Belle	37	
	Caen, Centre hospitalier regional universitaire de Caen	Prof. Farzin Beygui	33	9
	Nantes, Hopital Prive le Confluent	Dr Ashok Tirouvanziam	31	
	Montpellier, Clinique du Millenaire	Prof. Christophe Piot	30	4
	Caen, Hopital Prive Saint Martin	Dr Jean François Morelle	27	4
	Rouen, Clinique Saint-Hilaire	Dr Rene Koning	27	7
	Metz, Hopital de Mercy	Dr Mathieu Valla	24	3
	Dijon, GCIDB – Hopital Prive Dijon Bourgogne	Dr Philippe Brunel	23	5
	Nimes, CHU Caremeau	Dr Guillaume Cayla	18	4
	Creteil, Centre Hospitalier Universitaire Henri-Mondor	Prof. Emmanuel Teiger	12	2
	Paris, Hopital Universitaire Pitie-Salpetriere	Prof. Gilles Montalescot	10	2
	Paris, Hopital Europeen Georges-Pompidou	Prof. Christian Spaulding	9	1
	Saint-Denis, Centre Cardiologique du Nord	Dr Phillipe Guyon	3	
Germany			24	6
	Homburg, Saarland University	Prof. Felix Mahfoud	20	6
	Landshut, Landshut-Archdorf Krankenhaus	Dr Pyxaras, Stylianos	4	
Hungary			68	5

Country	Site name	Principal investigator	Patients randomised (n=4579)	Patients consented but not randomised (n=625)
	Budapest, Semmelweis University Heart and Vascular Center	Prof. Béla Merkely	46	5
	Szeged, Invasive Cardiology Unit University of Szeged	Dr Imre Ungi	22	
India			147	11
	Coimbatore, G Kuppuswamy Naidu Memorial Hospital	Dr Rajpal K Abhaichand	94	10
	Surat, Shri BD Mehta Mahavir Heart Institute	Dr Atul Damodar Abhyankar	33	
	Chennai, Apollo Hospitals, Chennai	Dr Sengottuvelu. G	13	1
	Chennai, Madras Medical Mission	Dr Ajit Mullasari. S	7	
Israel			100	33
	Safed, Ziv Medical Center, Cardiology Department	Dr Halabi Majdi	37	9
	Petach Tikva, Rabin MC	Prof. Ran Kornowski	34	11
	Haifa, Rambam Medical Center	Prof. Ariel Roguin----from 14th Oct 2018 Dr Yair Feld	16	6
	Jerusalem, Hadassah Ein Karem Medical Center	Prof. Chaim Lotan	13	7
Italy			276	37
	Rome, Policlinico Casilino	Dr Michael Donahue	99	6
	Vimercate, Ospedale di Vimercate	Dr Stefano Garducci	48	3
	Rozzano, Humanitas Research Hospital	Dr Bernhard Reimers	30	2
	Rome, Policlinico Umberto I	Dr Gennaro Sardella	20	2
	Milan, San Raffaele Hospital	Dr Antonio Colombo---from 20th June 2019 Dr Alaide Chieffo	12	1
	Catania, Ferrarotto Hospital	Prof. Corrado Tamburino	9	2
	Messina, AOU Policlinico Martino	Dr Giuseppe Andò	8	4
	Milan, Policlinico San Donato	Dr Luca Testa	8	4
	Milan, Sacco Hospital	Dr Maurizio Di Biasi	8	6
	Rome, Ospedale Sandro Pertini	Dr Alessandro Sciahbasi	8	3
	Caserta, Azienda Ospedaliera di Caserta Sant Anna e San Sebastiano	Prof.Dr Paolo Calabro	6	1
	Andria, Ospedale Lorenzo Bonomo	Dr Gianluigi Minervini	5	
	Cagliari, Azienda Ospedaliera Brotzu	Dr Bruno Loi	5	
	Milan, Centro Cardiologico Monzino IRCCS	Dr Franco Fabbiocchi	5	
	Milan, ASST Grande Ospedale Metropolitano Niguarda	Dr Jacopo Oreglia	4	3
	Treviglio, ASST Bergamo Ovest	Dr Paolo Sganzerla	1	

Country	Site name	Principal investigator	Patients randomised (n=4579)	Patients consented but not randomised (n=625)
Japan			188	17
	Toyoake, Fujita Health University Hospital	Prof. Yukio Ozaki	60	2
	Kokura, Fukuoka Kokura Memorial Hospital	Dr Kenji Ando	43	2
	Osaka, Osaka Police Hospital	Dr Yoshiharu Higuchi	22	4
	Tokyo, Sakakibara Heart Institute	Dr Mamoru Nanasato	13	1
	Kanagawa, St. Marianna University School of Medicine	Dr Yuki Ishibashi	11	1
	Gifu, Gifu Heart Center	Dr Hitoshi Matsuo	10	
	Nagoya, Japanese Red Cross Nagoya Daini Hospital	Dr Ruka Yoshida	8	2
	Ichinomiya, Ichinomiya municipal hospital	Dr Kiyokazu Shimizu	6	2
	Nagoya, Japanese Red Cross Nagoya	Dr Haruo Kamiya	4	2
	634 – Japan, Tokyo, St. Lukes International Hospital	Dr Nobuyuki Komiyama	4	1
	Nagakuteshi, Aichi Medical University Hospital	Dr Tetsuya Amano	3	
	Nagoya, Nagoya University Hospital	Dr Toyoaki Murohara	2	
	Sapporo, Sapporo Higashi Tokushukai Hospital	Dr Seiji Yamazaki	2	
Kingdom of Bahrain	Riffa, Bahrain Defence Force Hospital	Dr Husam Noor	7	1
Macedonia	Skopje, University Clinic of Cardiology	Dr Sasko Kedev	120	3
Poland			177	7
	Krakow, Institute Of Cardiology Jagiellonian University	Dr Jakub Podolec	69	4
	Poznan, Szpital Kliniczny Przemienienia Panskiego	Prof. Maciej Lesiak	50	1
	Wroclaw, 4 Wojskowy Szpital Kliniczny	Dr Krzysztof Reczuch	33	1
	Lubin, Miedziowe Centrum Zdrowia SA	Dr Adian Wlodarczak	18	1
	Krakow, University Hospital Krakow Poland	Prof. Dariusz Dudek	7	
Portugal	Lisbon, Hospital de Santa Maria	Dr Pedro Canas da Silva	1	
Saudi Arabia	King Fahd Armed Forces Hospital	Dr Mirvat Alasnag	16	1
Serbia			138	11
	Belgrade, Institute for Cardiovascular Disease Dedinje	Dr Ljupco Mangovski − from 17 April 2019 Dr Dragan Topic	67	4
	Belgrade, Clinical Center of Serbia	Prof. Goran Stankovic	61	7
	Sremska Kamenica, Institute of Cardiovascular Diseases	Dr Dragan Debeljacki	10	
Singapore			46	10

Country	Site name	Principal investigator	Patients randomised (n=4579)	Patients consented but not randomised (n=625)
	Singapore, Tan Tock Seng Hospital	Prof. Paul Ong Jau Lueng	38	10
	Singapore, KhooTeck Puat Hospital	Dr Syed Saqib Imran	8	
South Korea	Seoul, Asan Medical Center	Dr Park Seung-Jung	15	
Spain			196	10
	Huelva, Juan Ramon Jimenez Hospital	Dr José Francisco Diaz Fernandez	47	1
	Vigo, Alvaro Cunqueiro	Prof. Andrés Iniguez	40	2
	Barcelona, Hospital Vall Hebron	Dr Bruno Garcia del Blanco	27	
	Alicante, Hospital General Universitario de Alicante	Dr Vicente Mainar	19	2
	Madrid, Hospital 12 de Octubre	Dr Ivan Gomez Blazquez	17	
	El Palmar, Universitario Virgen de la Arrixaca	Dr Eduardo Pinar	15	1
	Madrid, Hospital Clinico San Carlos	Prof. Javier Escaned Barbosa	11	2
	Barcelona, Bellvitge University Hospital	Dr Joan Antoni Gomez Hospital	10	2
	Santander, Hospital Universitario Valdecilla	Dr Fermin Sainz	9	
	Majadahonda, Hospital Universitario Puerta de Hierro	Dr Javier Goicolea	1	
Sweden			8	
	Orebro, Orebro University Hospital	Dr Ole Fröbert	6	
	Gavle, Gavle Hospital	Dr Robert Kastberg	2	
Switzerland			499	111
	Bern, Inselspital	Dr Aris Moschovitis---from 20th Oct 2020 Prof. Stephan Windecker	308	61
	Liestal, Kantonsspital Baselland	Dr Gregor Leibundgut	68	14
	Lugano, Cardiocentro Ticino	Dr Giovanni Pedrazzini	31	9
	Geneva, University Hospital	Prof. Marco Roffi	29	13
	Bern, Lindenhofspital	Dr Ali Garachemani	28	3
	Zurich, University Hospital Zurich	Dr Patrick Siegrist	18	7
	Fribourg, HFR Hopital cantonal	Prof. Stéphane Cook	17	4
Netherlands			539	122
	Rotterdam, Maasstad Ziekenhuis	Dr Peter Smits	233	79
	Terneuzen, Zorgsaam	Dr Al Mafragi	87	5
	Emmen, Treant Zorggroep	PI Dr Jessurun---from 1st July 2020 Dr Ruifrok	67	9
	Eindhoven, Catharina Ziekenhuis	Dr Pim Tonino	54	10
	Arnhem, Rijnstate Ziekenhuis	Dr Peter Danse	29	8

Country	Site name	Principal investigator	Patients randomised (n=4579)	Patients consented but not randomised (n=625)
	Hertogenbosch, Jeroen Bosch Ziekenhuis	Dr J. Polad	21	3
	Dordrecht, Albert Schweitzer Ziekenhuis	Dr Floris Kauer	20	6
	Enschede, Medisch Spectrum Twente	Dr Clemens von Birgelen	19	
	Nieuwegein, Antonius Ziekenhuis Nieuwegein	Dr Jurrien ten Berg	5	1
	Breda, Amphia Ziekenhuis	Dr Sander Ijsselmuiden	3	
	Den Haag, Hagahospital	Dr Samer Somi	1	1
United Kingdom			279	48
	Bristol, Bristol Heart Institute	Dr Tom Johnson	55	13
	Worcester, Worcestershire Royal Hospital	Dr Helen Routledge	43	8
	Brighton, Brighton & Sussex University Hospitals Trust	Dr David Hildick-Smith	40	3
	Bournemouth, Royal Bournemouth Hospital	Dr Jehangir Din	34	7
	Wolverhampton, Heart and Lung Centre – New Cross Hospital	Dr Shahzad Munir	22	6
	Blackburn, Royal Blackburn Hospital	Dr John McDonald	20	1
	Stevenage, Lee Haynes Research Institute, Lister Hospital	Dr Neville Kukreja	20	1
	Stoke on Trent, Royal Stoke University Hospital	Prof. Mamas Mamas	20	5
	Newcastle upon Tyne, Freeman Hospital	Dr Rajiv Das	13	1
	Manchester, Wythenshawe Hospital	Dr Hussain Contractor	8	3
	Derry, Altnagelvin Hospital	Dr Aaron Peace	2	
	London, St. George's Hospitals	Dr Rupert Williams	2	
Vietnam	Vietnam National Heart Institute – Bach Mai Hospital Hanoi	Prof. Nguyen Ngoc Quang	25	2

Additional information on the methods

1. Criteria for High Bleeding Risk

Post-percutaneous coronary intervention (PCI), patients are at high bleeding risk if at least one of the following criteria applies:

1. Clinical indication for treatment with oral anticoagulant (OAC) for at least 12 months.
2. Recent (<12 months) nonaccess site bleeding episode(s) that required medical attention (i.e. actionable bleeding).
3. Previous bleeding episode(s) that required hospitalization if the underlying cause had not been definitively treated (i.e. surgical removal of the bleeding source).
4. Age ≥75 years.
5. Systemic conditions associated with an increased bleeding risk (e.g. hematological disorders, including a history of current thrombocytopenia defined as a platelet count <100.00/ mm^3 (<100 × 10^9/L) or any known coagulation disorder associated with increased bleeding risk.
6. Documented anemia, defined as repeated hemoglobin levels <11 g/dL or transfusion during the 4 weeks before inclusion.
7. Need for chronic treatment with steroids or nonsteroidal anti-inflammatory drugs.
8. Diagnosed malignancy (other than skin) considered at high bleeding risk including gastrointestinal, genitourethral/renal and pulmonary.
9. Stroke at any time or transient ischemic attack in the previous 6 months.
10. PRECISE-DAPT score ≥25.

2. Inclusion and Exclusion Criteria

Inclusion Criteria

Inclusion criteria after index PCI

1. Age ≥18 years
2. At least one high bleeding risk criterion (listed above)
3. All coronary lesions successfully treated with Ultimaster stent
4. Free of any flow-limiting angiographic complications that required prolonged dual antiplatelet therapy (DAPT) duration based on operator's decision
5. All stages of PCI were complete and no further PCI was planned

Inclusion criteria at 1-month randomization visit (30−44 days after qualifying index PCI)

1. At least one high bleeding risk criterion (listed above) or on the basis of post-PCI actionable nonaccess-site related bleeding episode
2. Uneventful 30-day clinical course (i.e. freedom from any new episode of acute coronary syndrome, symptomatic restenosis, stent thrombosis, stroke, any revascularization requiring prolonged DAPT)
3. If not on OAC:
   a) Patient was on DAPT regimen of aspirin and a $P2Y_{12}$ inhibitor;
   b) Patient with one type of $P2Y_{12}$ inhibitor for at least 7 days
4. If on OAC:
   a) Patient was on the same type of OAC for at least 7 days;
   b) Patient was on clopidogrel for at least 7 days

Exclusion Criteria

Patients were not eligible if any of the following applied:

1. Treated with stent other than Ultimaster stent within 6 months prior to index PCI
2. Treated for in-stent restenosis or stent thrombosis at index PCI or within 6 months before
3. Treated with a bioresorbable scaffold at any time prior to index procedure
4. Incapable of providing written informed consent
5. Under judicial protection, tutorship or curatorship
6. Unable to understand and follow study-related instructions or unable to comply with study protocol
7. Active bleeding requiring medical attention (Bleeding Academic Research Consortium [BARC] ≥2) on randomization visit
8. Life expectancy less than 1 year
9. Known hypersensitivity or allergy to aspirin, clopidogrel, ticagrelor, prasugrel, cobalt chromium or sirolimus
10. Any planned and anticipated PCI
11. Participation in another trial
12. Pregnant or breastfeeding women

3. Treatment Regimen

Patients were treated according to the randomized regimen from the day of randomization until 11 months after randomization (12 months after the index procedure). After 11 months post randomization, antiplatelet therapy was at the discretion of treating physician.

3.1. Abbreviated DAPT regimen

In patients not on OAC: DAPT was discontinued, and a single antiplatelet agent (SAPT) was continued until at least 11 months post randomization (i.e. 12 months after index PCI).

In patients on OAC: DAPT was discontinued. Either aspirin or clopidogrel was continued until 5 months post randomization (i.e. 6 months after index PCI). OAC was continued until at least 11 months post randomization (i.e. 12 months after index PCI).

3.2. Standard DAPT regimen

In patients not on OAC: Aspirin was continued until at least 11 months post randomization (i.e. 12 months after index PCI). The P2Y12 inhibitor being taken at the time of randomization was continued for at least 5 months and up to 11 months post randomization (i.e. 12 months after index PCI).

In patients on OAC: Aspirin and clopidogrel were continued for at least 2 months (i.e. 3 months after index PCI) and up to 11 months post randomization (i.e. 12 months after index PCI). Thereafter, either aspirin or clopidogrel was continued up to 11 months post randomization (i.e. 12 months after index PCI). OAC was continued until at least 11 months post randomization (i.e. 12 months after index PCI).

The rationale for mandating clopidogrel as the only acceptable P2Y12 inhibitor in the OAC population in both study arms came from the absence of safety and efficacy data regarding the combination of ticagrelor or prasugrel with aspirin and OAC (as patients requiring OAC were excluded from approval RCT) and a recommendation of Class III (i.e. not indicated) in the European guidelines.

3.3. Implementation of randomized study regimens

Study regimens were implemented by regular drug prescription as described above. The investigators provided the necessary prescription to the study participants. The following are recommended according to the current guidelines and local practice.

- Aspirin is prescribed at the standard dose of at least 75 mg/day and up to 162 mg/day.
- Clopidogrel is prescribed in standard dose of 75 mg once daily.
- Prasugrel is prescribed at the standard dose of 10 mg/day or 5 mg/day in patients weighing less than 60 kg or who are over 75 years old. In regions where other standard dose exists (i.e. Japan), prasugrel dosage is adjusted according to the locally approved dose.
- Ticagrelor is prescribed at the standard dose of 180 mg/day (90 mg b.i.d.).
- Vitamin K antagonist is dosed to keep the international normalized ratio within the guideline range.
- Nonvitamin K oral antagonist oral anticoagulants (NOAC; rivaroxaban, edoxaban, dabigatran and apixaban) are given in locally approved doses.
- Switching from a vitamin K antagonist to NOAC or vice-versa is not allowed unless there are clinical and well documented reasons for doing so. Similarly, switching from a NOAC to a VKA during the course of the study is not allowed, unless dictated by a clinical and documented reason (e.g. change in renal function during the course of the investigation), which will be captured in the eCRF.

Prescribed units of aspirin, clopidogrel, prasugrel, ticagrelor and OAC were recorded in the eCRF. Patients are queried on general drug adherence.

4. Outcome Definitions

4.1. Death

All deaths were categorized as cardiovascular, noncardiovascular, or undetermined based on the definitions below.

4.1.1. Cardiovascular death

Cardiovascular death was defined as death resulting from an acute myocardial infarction, sudden cardiac death, death due to heart failure, death due to stroke, death due to cardiovascular procedures, death due to cardiovascular hemorrhage, and death due to other cardiovascular causes.

4.1.2. Death due to acute myocardial infarction

Death due to acute myocardial infarction was death by any mechanism (arrhythmia, heart failure, low output) within 30 days after a myocardial infarction related to the immediate consequences of the myocardial infarction, such as progressive congestive heart failure (CHF), inadequate cardiac output, or refractory arrhythmia. If these events occurred after a "break" (e.g. a CHF- and arrhythmia-free period of at least a week), they were designated by the immediate cause, even though the myocardial infarction may have increased the risk of that event (e.g. late arrhythmic death becomes more likely after an acute myocardial infarction). The acute myocardial infarction was verified to the extent possible by the diagnostic criteria outlined for acute myocardial infarction or by autopsy findings showing recent myocardial infarction or recent coronary thrombus. Sudden cardiac death, if accompanied by symptoms suggestive of myocardial ischemia, new ST elevation, new left bundle branch block (LBBB), or evidence of fresh thrombus by coronary angiography and/or at autopsy should be considered death resulting from an acute myocardial infarction, even if death occurs before blood samples or 12-lead electrocardiogram (ECG) could be obtained, or at a time before the appearance of cardiac biomarkers in the blood. Death resulting from a procedure to treat a myocardial infarction (i.e. PCI, coronary artery bypass graft surgery [CABG]), or to treat a complication resulting from myocardial infarction, should also be considered death due to acute myocardial infarction. Death resulting from an elective coronary procedure to treat myocardial ischemia (i.e. chronic stable angina) or death due to a myocardial infarction that occurred as a direct consequence of a cardiovascular investigation/procedure/operation should be considered as a death due to a cardiovascular procedure.

4.1.3. Sudden cardiac death

Sudden cardiac death was death that occurred unexpectedly, not following an acute myocardial infarction, and included the following:

- Death witnessed and occurring without new or worsening symptoms.
- Death witnessed within 60 min of the onset of new or worsening cardiac symptoms, unless documented (i.e. by ECG or other objective) to be due to acute myocardial infarction.
- Death witnessed and attributed to an identified arrhythmia (e.g. captured on an ECG recording, witnessed on a monitor, or unwitnessed but found on implantable cardioverter-defibrillator review).
- Death after unsuccessful resuscitation from cardiac arrest. Death after successful resuscitation from cardiac arrest and without identification of a noncardiac etiology.
- Unwitnessed death without other cause of death (information regarding the patient's clinical status preceding death should be provided, if available).

4.1.3.1. GENERAL CONSIDERATIONS

A subject seen alive and clinically stable 24 h prior to being found dead without any evidence or information of a specific cause of death was classified as "sudden cardiac death." Typical scenarios included:

- Subject well the previous day but found dead in bed the next day
- Subject found dead at home on the couch with the television on
- Deaths for which there was no information beyond "Patient found dead at home" may be classified as "death due to other cardiovascular causes".

4.1.4. Death due to heart failure or cardiogenic shock

Death due to congestive heart failure referred to a death in association with clinically worsening symptoms and/or signs of heart failure not following an acute myocardial infarction. Deaths due to heart failure could have various etiologies, including single or recurrent myocardial infarctions, ischemic or nonischemic cardiomyopathy, hypertension, or valvular disease. Cardiogenic shock not occurring in the context of an acute myocardial infarction or as the consequence of an arrhythmia occurring in the absence of worsening heart failure was defined as systolic blood pressure (SBP) <90 mm Hg for greater than 1 hour, not responsive to fluid resuscitation and/or heart rate correction, and felt to be secondary to cardiac dysfunction and associated with at least one of the following signs of hypoperfusion:

- Cool, clammy skin or
- Oliguria (urine output <30 mL/h) or
- Altered sensorium or
- Cardiac index <2.2 L/min/m^2

- Cardiogenic shock could also be defined if SBP <90 mm Hg and increased to ≥90 mm Hg in less than 1 hour with positive inotropic or vasopressor agents alone and/or with mechanical support.

4.1.5. Death due to stroke

Death due to stroke referred to death after a stroke that was either a direct consequence of the stroke or a complication of the stroke. Acute stroke was verified to the extent possible by the diagnostic criteria outlined for stroke.

4.1.6. Death due to cardiovascular procedures

Death due to cardiovascular procedures referred to death caused by the immediate complications of a cardiac procedure and excluded death resulting from procedures to treat an acute myocardial infarction or the complications resulting from an acute myocardial infarction.

4.1.7. Death due to cardiovascular hemorrhage

Death due to cardiovascular hemorrhage referred to death related to hemorrhage such as a nonstroke intracranial hemorrhage, nonprocedural or nontraumatic vascular rupture (e.g. aortic aneurysm), or hemorrhage causing cardiac tamponade.

4.1.8. Death due to other cardiovascular causes

Death due to other cardiovascular causes referred to a cardiovascular death not included in the above categories (e.g. pulmonary embolism or peripheral artery disease).

4.1.9. Noncardiovascular death

Noncardiovascular death was defined as any death that was not thought to be due to a cardiovascular cause. The following categories may be collected.

4.1.9.1. NONMALIGNANT CAUSES

- Pulmonary
- Renal
- Gastrointestinal
- Hepatobiliary
- Pancreatic
- Infection (includes sepsis)
- Noninfectious (e.g. systemic inflammatory response syndrome)
- Hemorrhage*, excluding hemorrhagic strokes and bleeding in the setting of coronary revascularization
- Noncardiovascular procedure or surgery
- Accidental (e.g. physical accidents or drug overdose) or trauma
- Suicide
- Prescription drug error (e.g. prescribed drug overdose, use of inappropriate drug, or drug-drug interaction)
- Neurological process that was not a stroke or hemorrhage
- Other noncardiovascular, specify: _____

**Examples: Death due to gastrointestinal bleeding was not considered a cardiovascular death. Death due to retroperitoneal hematoma following PCI was considered cardiovascular death. Death due to intracerebral hemorrhage was considered cardiovascular death.*

4.1.9.2. MALIGNANT CAUSES

Death from a malignant cause was that resulting directly from the cancer, or death resulting from a complication of the cancer (e.g. infection, complication of surgery / chemotherapy / radiotherapy), or death resulting from withdrawal of other therapies because of concerns relating to the poor prognosis associated with the cancer.

Cancer deaths that arose from cancers that were present prior to randomization or which developed subsequently were further classified (worsening prior malignancy; new malignancy).

4.1.10. Undetermined cause of death

Undetermined cause of death referred to a death not attributable to one of the above categories of cardiovascular death or to a noncardiovascular cause, due to absence of any information (e.g. the only available information is “patient died”). The use

of this category of death was discouraged and should have only applied to a minimal number of cases when no information at all on the circumstances of death were available (i.e. found on obituary of local newspaper). In all circumstances the reviewer used all available information to attribute to one of the categories based on best clinical judgment.

For each death event an assessment was made as to whether the event was caused (on the basis of the totality of the evidence) by a bleeding (i.e. a fatal bleeding occurred) or not.

4.2. Myocardial Infarction

For the primary analysis, the myocardial infarction outcome was defined based on the Third Universal Definition of myocardial infarction with the exception of periprocedural myocardial infarction after PCI, which was defined according to the Society for Cardiovascular Angiography and Intervention (SCAI) definition. For secondary analyses, PCI-related myocardial infarction according to the Third Universal Definition (type 4a) was also adjudicated.

4.2.1. Spontaneous myocardial infarction (>48 h after intervention, myocardial infarction type 1)

Symptoms suggestive of ischemia/infarction in association with ECG, cardiac biomarker, or pathologic evidence of infarction were as follows:

- Detection of a rise and/or fall of cardiac biomarker values (preferably cardiac troponin T or I) with at least one value above the 99th percentile upper reference limit (URL) and with at least one of the following:
    - Symptoms of ischemia
    - New or presumed new significant ST segment-T wave (ST-T) changes or new LBBB
    - Development of new Q waves in the ECG
    - Evidence of new loss of viable myocardium or new regional wall motion abnormality
    - Identification of an intracoronary thrombus by angiography or autopsy

Spontaneous myocardial infarction typically occurs after the periprocedural period and may be secondary to late stent complications or progression of native disease (e.g. nonculprit lesion plaque rupture). Performance of ECG and angiography supports adjudication to either a target or nontarget vessel or lesion in most cases.

4.2.2. Type 2 myocardial infarction

In instances of myocardial injury with necrosis where a condition other than coronary artery disease (CAD) contributes to an imbalance between myocardial oxygen supply and/or demand, e.g. coronary endothelial dysfunction, coronary artery spasm, coronary embolism, tachy/bradyarrhythmias, anemia, respiratory failure, hypotension, and hypertension with or without left ventricular hypertrophy.

4.2.3. Type 3 myocardial infarction

Cardiac death with symptoms suggestive of myocardial ischemia and presumed new ischemic ECG changes or new LBBB, but death occurred before cardiac biomarkers were obtained, or before cardiac biomarker values would be increased.

4.2.4. Type 4a myocardial infarction (not used for primary analysis, see section 4.8 for primary definition of periprocedural myocardial infarction)

Type 4a myocardial infarction was defined by elevation of cardiac troponin (cTn) values (>5 × URL) occurring within 48 h of the procedure in patients with normal baseline values (≤URL) or a rise of cTn values >20% if the baseline values are elevated and are stable or falling. In addition, at least one of the following was required:

- Symptoms suggestive of myocardial ischemia
- New ischemic ECG changes
- Angiographic findings consistent with a procedural complication
- Imaging demonstration of new loss of viable myocardium or new regional wall motion abnormality.

4.2.5. Type 4b myocardial infarction

Type 4b myocardial infarction was defined as stent thrombosis associated with myocardial infarction when detected by coronary angiography or autopsy in the setting of evidence of myocardial ischemia and with a rise and/or fall of cardiac biomarker values with at least one value above the URL.

4.2.6. Type 4c myocardial infarction

Type 4c myocardial infarction was defined as spontaneous myocardial infarction where a restenosis was the only angiographic explanation.

4.2.7. Type 5 myocardial infarction

4.2.7.1. CORONARY ARTERY BYPASS GRAFTING-RELATED MYOCARDIAL INFARCTION

Coronary artery bypass grafting (CABG) related myocardial infarction was defined by elevation of troponin values (>10 × URL) occurring within 48 h of the procedure in patients with normal baseline cTn values (≤URL). In addition, at least one of the following was required:

- New pathological Q waves or new LBBB
- Angiographic documented new graft or new native coronary artery occlusion
- Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.

4.2.8. Periprocedural myocardial infarction after PCI (within 48 h of PCI)

Periprocedural myocardial infarction was defined based on the SCAI definitions as follows:

1) In patients with normal baseline creatine kinase-MB (CK-MB): The peak CK-MB measured within 48 h of the procedure rises to ≥10 × the local laboratory ULN, or to ≥5 × ULN with new pathologic Q-waves in ≥2 contiguous leads or new persistent LBBB, OR in the absence of CK-MB measurements and a normal baseline cTn, a cTn (I or T) level measured within 48 h of the PCI rises to ≥70 × the local laboratory ULN, or ≥35 × ULN with new pathologic Q-waves in ≥2 contiguous leads or new persistent LBBB.
2) In patients with elevated baseline CK-MB (or cTn) in whom the biomarker levels are stable or falling: The CK-MB (or cTn) rises by an absolute increment equal to those levels recommended above from the most recent preprocedure level.
3) In patients with elevated CK-MB (or cTn) in whom the biomarker levels have not been shown to be stable or falling: The CK-MB (or cTn) rises by an absolute increment equal to those levels recommended above plus new ST-segment elevation or depression plus signs consistent with a clinically relevant myocardial infarction, such as new onset or worsening heart failure or sustained hypotension.

4.2.9. Target-vessel vs. nontarget-vessel myocardial infarction

Any myocardial infarction not clearly attributable to a nontarget vessel was considered as target-vessel myocardial infarction.

4.3. Stent Thrombosis

Stent thrombosis was defined by the Academic Research Consortium as follows:

4.3.1. Definite stent thrombosis

Definite stent thrombosis was considered to have occurred by either angiographic or pathological confirmation:

a. *Angiographic confirmation of stent thrombosis (the incidental angiographic documentation of stent occlusion in the absence of clinical signs or symptoms was not considered a confirmed stent thrombosis ([silent occlusion])*

The presence of an intracoronary thrombus that originates in the stent or in the segment 5 mm proximal or distal to the stent and presence of at least 1 of the following criteria within a 48-h time window:

- Acute onset of ischemic symptoms at rest
- New ischemic ECG changes that suggest acute ischemia
- Typical rise and fall in cardiac biomarkers (refer to definition of spontaneous myocardial infarction: Troponin or CK-MB >99th percentile of URL)
- Nonocclusive thrombus. Intracoronary thrombus was defined as a (spheric, ovoid, or irregular) noncalcified filling defect or lucency surrounded by contrast material (on 3 sides or within a coronary stenosis) seen in multiple projections, or persistence of contrast material within the lumen, or a visible embolisation of intraluminal material downstream
- Occlusive thrombus Thrombolysis In Myocardial Infarction (TIMI) 0 or TIMI 1 intrastent or proximal to a stent up to the most adjacent proximal side branch or main branch (if originates from the side branch)

b. Pathological confirmation of stent thrombosis

- Evidence of recent thrombus within the stent determined at autopsy or via examination of tissue retrieved following thrombectomy

4.3.2. Probable stent thrombosis

Clinical definition of probable stent thrombosis was considered to have occurred after intracoronary stenting in the following cases:

- Any unexplained death within the first 30 days.
- Irrespective of the time after the index procedure, any myocardial infarction related to documented acute ischemia in the territory of the implanted stent without angiographic confirmation of stent thrombosis and in the absence of any other obvious cause.

4.3.3. Possible stent thrombosis

Clinical definition of possible stent thrombosis was considered to have occurred with any unexplained death from 30 days following intracoronary stenting until end of trial follow-up.

4.4. Stroke

Stroke was defined as an acute episode of focal or global neurological dysfunction caused by central nervous system (CNS) vascular injury as a result of hemorrhage or infarction. CNS included brain, spinal cord and retina. Stroke was defined as follows.

4.4.1. Ischemic stroke

Ischemic stroke was defined as an acute episode of focal cerebral, spinal, or retinal dysfunction caused by CNS infarction. Evidence of infarction was defined as "Pathological, imaging, or other objective evidence of acute cerebral, spinal cord, or retinal focal ischemic injury in a defined vascular distribution; or in the absence of the above (i.e. imaging or autopsy unavailable), clinical evidence of cerebral, spinal cord, or retinal focal ischemic injury was based on symptoms persisting for ≥24 h or until death, and other etiologies excluded. Hemorrhagic infarction, defined as a parenchymal hemorrhage after CNS infarction, was considered an ischemic stroke

4.4.2. Cerebral hemorrhage

Hemorrhages in the CNS were classified as stroke if they were nontraumatic, caused by a vascular event, and resulted in injury to the CNS. In contrast, traumatic hemorrhages were not characterized as stroke. Subdural hematoma was not classified as a stroke. The diagnoses included in this section were intracerebral hemorrhage (intraparenchymal and intraventricular) and subarachnoid hemorrhage (both aneurysmal and nonaneurysmal).

4.4.3. Stroke caused by intracerebral hemorrhage

Rapidly developing clinical signs of neurological dysfunction (focal or global) attributable to a focal collection of blood within the brain parenchyma or ventricular system that was not caused by trauma.

4.4.4. Stroke caused by subarachnoid hemorrhage

Rapidly developing signs of neurological dysfunction (focal or global) and/or headache because of bleeding into the subarachnoid space (the space between the arachnoid membrane and the pia mater of the brain or spinal cord), which was not caused by trauma. Hemorrhages could be further classified according to location (example, supratentorial, subtentorial, etc.)

4.4.5. Stroke not otherwise specified

An episode of acute neurological dysfunction presumed to be caused by ischemia or hemorrhage, persisting ≥24 h or until death, but without sufficient evidence to be classified as one of the above.

4.5. Bleeding

All potential bleeding events were primarily adjudicated according to Bleeding Academic Research Consortium (BARC) classification.

Type 0	No bleeding
Type 1	Bleeding that is not actionable and does not cause the patient to seek unscheduled performance of studies, hospitalization, or treatment by a health care professional. May include episodes leading to self-discontinuation of medical therapy by the patient, without consulting a health care professional.
Type 2	Any overt, actionable sign of hemorrhage (e.g. more bleeding than would be expected for a clinical circumstance; including bleeding found by imaging alone) that does not fit the criteria for Types 3, 4, or 5 but does meet at least one of the following criteria: Requiring nonsurgical, medical intervention by a health care professional Leading to hospitalization of increased level of care

	Prompting evaluation
Type 3a	Overt bleeding plus hemoglobin drop of 3 to <5* g/dL (provided hemoglobin drop is related to bleed) Any transfusion with overt bleeding
Type 3b	Overt bleeding plus hemoglobin drop ≥5* g/dL (provided hemoglobin drop is related to bleed) Cardiac tamponade Bleeding requiring surgical intervention for control (excluding dental / nasal / skin / hemorrhoid) Bleeding requiring intravenous vasoactive agents
Type 3c	Intracranial hemorrhage (does not include microbleeds or hemorrhagic transformation; does include intraspinal) Subcategories: confirmed by autopsy or imaging or lumbar puncture Intra-ocular bleed compromising vision
Type 4	CABG-related bleeding Perioperative intracranial bleeding within 48 h Reoperation following closure of sternotomy for the purpose of controlling bleeding Transfusion of ≥5 units of whole blood or packed red blood cells within 48 hour period† Chest tube output ≥2 L within a 24 hour period
Type 5a	Probable fatal bleeding; no autopsy or imaging confirmation, but clinically suspicious
Type 5b	Definite fatal bleeding: overt bleeding or autopsy or imaging confirmation

Platelet transfusions were recorded and reported, and were not included in these definitions until further information was obtained about the relationship to outcomes.

*Corrected for transfusion (1 unit packed red blood cells or 1 unit whole blood=1g/dL hemoglobin).

†Cell saver products were not counted.

Supplementary Table 1. Details on DAPT and SAPT at 1-, 3-, 6- and 12-month study visits in CKD versus non-CKD patients stratified by the randomly allocated antiplatelet regimen.

	CKD			Non-CKD		
	Abbreviated DAPT (n=711)	**Standard DAPT (n=717)**	**p-value**	**Abbreviated DAPT (n=1584)**	**Standard DAPT (n=1567)**	**p-value**
at 1 month visit (before randomisation)						
DAPT	n = 711, 708 (99.6%)	n = 717, 712 (99.3%)	0.726	n = 1584, 1573 (99.3%)	n = 1567, 1560 (99.6%)	0.480
Clopidogrel	n = 711, 563 (79.2%)	n = 717, 561 (78.2%)	0.698	n = 1584, 1254 (79.2%)	n = 1567, 1232 (78.6%)	0.727
Prasugrel	n = 717, 11 (1.5%)	n = 717, 12 (1.7%)	1.000	n = 1584, 48 (3.0%)	n = 1567, 44 (2.8%)	0.751
Ticagrelor	n = 711, 134 (18.8%)	n = 717, 139 (19.4%)	0.840	n = 1584, 271 (17.1%)	n = 1567, 284 (18.1%)	0.483
SAPT	n = 717, 3 (0.4%)	n = 717, 5 (0.7%)	0.726	n = 1584, 11 (0.7%)	n = 1567, 7 (0.4%)	0.480
Acetylsalicylic acid	n = 717, 1 (0.1%)	n = 717, 1 (0.1%)	1.000	n = 1584, 2 (0.1%)	n = 1567, 0 (0.0%)	0.500
Clopidogrel	n = 717, 2 (0.3%)	n = 717, 4 (0.6%)	0.687	n = 1584, 9 (0.6%)	n = 1567, 7 (0.4%)	0.803
Prasugrel	n = 717, 0 (0.0%)	n = 717, 0 (0.0%)	-	n = 1584, 0 (0.0%)	n = 1567, 0 (0.0%)	-
Ticagrelor	n = 717, 0 (0.0%)	n = 717, 0 (0.0%)	-	n = 1584, 0 (0.0%)	n = 1567, 0 (0.0%)	-
at 1 month visit (after randomisation)						
DAPT	n = 711, 12 (1.7%)	n = 717, 714 (99.6%)	<0.001	n = 1584, 40 (2.5%)	n = 1567, 1558 (99.4%)	<0.001
Clopidogrel	n = 711, 10 (1.4%)	n = 717, 565 (78.8%)	<0.001	n = 1584, 29 (1.8%)	n = 1567, 1233 (78.7%)	<0.001
Prasugrel	n = 711, 0 (0.0%)	n = 717, 12 (1.7%)	<0.001	n = 1584, 1 (0.1%)	n = 1567, 43 (2.7%)	<0.001
Ticagrelor	n = 711, 2 (0.3%)	n = 717, 137 (19.1%)	<0.001	n = 1584, 10 (0.6%)	n = 1567, 282 (18.0%)	<0.001

SAPT	n = 711, 696 (97.9%)	n = 717, 3 (0.4%)	<0.001	n = 1584, 1538 (97.1%)	n = 1567, 6 (0.4%)	<0.001
Acetylsalicylic acid	n = 711, 207 (29.1%)	n = 717, 1 (0.1%)	<0.001	n = 1584, 453 (28.6%)	n = 1567, 0 (0.0%)	<0.001
Clopidogrel	n = 711, 383 (53.9%)	n = 717, 2 (0.3%)	<0.001	n = 1584, 853 (53.9%)	n = 1567, 6 (0.4%)	<0.001
Prasugrel	n = 711, 6 (0.8%)	n = 717, 0 (0.0%)	0.015	n = 1584, 21 (1.3%)	n = 1567, 0 (0.0%)	<0.001
Ticagrelor	n = 711, 100 (14.1%)	n = 717, 0 (0.0%)	<0.001	n = 1584, 211 (13.3%)	n = 1567, 0 (0.0%)	<0.001
at 3 months visit						
DAPT	n = 702, 23 (3.3%)	n = 708, 611 (86.3%)	<0.001	n = 1560, 48 (3.1%)	n = 1546, 1326 (85.8%)	<0.001
Clopidogrel	n = 702, 18 (2.6%)	n = 708, 471 (66.5%)	<0.001	n = 1560, 38 (2.4%)	n = 1546, 1016 (65.7%)	<0.001
Prasugrel	n = 702, 0 (0.0%)	n = 708, 12 (1.7%)	<0.001	n = 1560, 1 (0.1%)	n = 1546, 44 (2.8%)	<0.001
Ticagrelor	n = 702, 5 (0.7%)	n = 708, 128 (18.1%)	<0.001	n = 1560, 9 (0.6%)	n = 1546, 266 (17.2%)	<0.001
SAPT	n = 702, 674 (96.0%)	n = 708, 92 (13.0%)	<0.001	n = 1560, 1506 (96.5%)	n = 1546, 218 (14.1%)	<0.001
Acetylsalicylic acid	n = 702, 198 (28.2%)	n = 708, 29 (4.1%)	<0.001	n = 1560, 447 (28.7%)	n = 1546, 59 (3.8%)	<0.001
Clopidogrel	n = 702, 376 (53.6%)	n = 708, 61 (8.6%)	<0.001	n = 1560, 842 (54.0%)	n = 1546, 159 (10.3%)	<0.001
Prasugrel	n = 702, 6 (0.9%)	n = 708, 0 (0.0%)	0,015	n = 1560, 19 (1.2%)	n = 1546, 0 (0.0%)	<0.001
Ticagrelor	n = 702, 94 (13.4%)	n = 708, 2 (0.3%)	<0.001	n = 1560, 199 (12.8%)	n = 1546, 0 (0.0%)	<0.001
at 6 months visit						
DAPT	n = 686, 21 (3.1%)	n = 691, 468 (67.7%)	<0.001	n = 1544, 49 (3.2%)	n = 1529, 904 (59.1%)	<0.001
Clopidogrel	n = 686, 16 (2.3%)	n = 691, 343 (49.6%)	<0.001	n = 1544, 42 (2.7%)	n = 1529, 624 (40.8%)	<0.001
Prasugrel	n = 686, 0 (0.0%)	n = 691, 11 (1.6%)	0,001	n = 1544, 0 (0.0%)	n = 1529, 42 (2.7%)	<0.001
Ticagrelor	n = 686, 5 (0.7%)	n = 691, 114 (16.5%)	<0.001	n = 1544, 7 (0.5%)	n = 1529, 238 (15.6%)	<0.001

SAPT	n = 686, 627 (91.4%)	n = 691, 217 (31.4%)	<0.001	n = 1544, 1346 (87.2%)	n = 1529, 609 (39.8%)	<0.001
Acetylsalicylic acid	n = 686, 196 (28.6%)	n = 691, 79 (11.4%)	<0.001	n = 1544, 412 (26.7%)	n = 1529, 242 (15.8%)	<0.001
Clopidogrel	n = 686, 337 (49.1%)	n = 691, 134 (19.4%)	<0.001	n = 1544, 722 (46.8%)	n = 1529, 365 (23.9%)	<0.001
Prasugrel	n = 686, 5 (0.7%)	n = 691, 0 (0.0%)	0,030	n = 1544, 22 (1.4%)	n = 1529, 0 (0.0%)	<0.001
Ticagrelor	n = 686, 89 (13.0%)	n = 691, 4 (0.6%)	<0.001	n = 1544, 191 (12.4%)	n = 1529, 2 (0.1%)	<0.001
at 12 months visit						
DAPT	n = 660, 29 (4.4%)	n = 659, 262 (39.8%)	<0.001	n = 1525, 72 (4.7%)	n = 1508, 508 (33.7%)	<0.001
Clopidogrel	n = 660, 21 (3.2%)	n = 659, 192 (29.1%)	<0.001	n = 1525, 58 (3.8%)	n = 1508, 346 (22.9%)	<0.001
Prasugrel	n = 660, 0 (0.0%)	n = 659, 7 (1.1%)	0,008	n = 1525, 1 (0.1%)	n = 1508, 15 (1.0%)	<0.001
Ticagrelor	n = 660, 8 (1.2%)	n = 659, 63 (9.6%)	<0.001	n = 1525, 13 (0.9%)	n = 1508, 147 (9.7%)	<0.001
SAPT	n = 660, 427 (64.7%)	n = 659, 346 (52.5%)	<0.001	n = 1525, 945 (62.0%)	n = 1508, 839 (55.6%)	<0.001
Acetylsalicylic acid	n = 660, 190 (28.8%)	n = 659, 231 (35.1%)	0,015	n = 1525, 421 (27.6%)	n = 1508, 545 (36.1%)	<0.001
Clopidogrel	n = 660, 168 (25.5%)	n = 659, 114 (17.3%)	<0.001	n = 1525, 375 (24.6%)	n = 1508, 290 (19.2%)	<0.001
Prasugrel	n = 660, 4 (0.6%)	n = 659, 0 (0.0%)	0,124	n = 1525, 22 (1.4%)	n = 1508, 0 (0.0%)	<0.001
Ticagrelor	n = 660, 65 (9.8%)	n = 659, 1 (0.2%)	<0.001	n = 1525, 127 (8.3%)	n = 1508, 4 (0.3%)	<0.001

Abbreviations: CKD, chronic kidney disease; DAPT, dual antiplatelet therapy; SAPT, single antiplatelet therapy.

Data are n (%) or n/N (%).

Patients switched to routine care around 12 months visit post-qualifying PCI; switching was allowed inside a 14-day window.

Supplementary Table 2. Baseline characteristics among CKD and non-CKD patients.

	CKD (n=1428)	Non-CKD (n=3151)	p-value
Age, years	77.35 ± 8.09	75.44 ± 8.95	<0.001
Male sex	904 (63.3%)	2267 (71.9%)	<0.001
BMI	27.73 ± 4.80	27.17 ± 4.66	<0.001
Family history of CAD	320 (22.4%)	789 (25.0%)	0.058
Arterial hypertension	1206 (84.5%)	2347 (74.5%)	<0.001
Hyperlipidemia	961 (67.3%)	2136 (67.8%)	0.759
Current smoking	113 (7.9%)	301 (9.6%)	0.084
LVEF	51.24 ± 12.13	54.13 ± 11.24	<0.001
Prior MI	318 (22.3%)	546 (17.3%)	<0.001
Prior PCI	401 (28.1%)	787 (25.0%)	0.029
Prior cerebrovascular event	187 (13.1%)	383 (12.2%)	0.385
Prior CABG	129 (9.0%)	212 (6.7%)	0.007
Diabetes mellitus	555 (38.9%)	983 (31.2%)	<0.001
Active cancer	63 (4.4%)	173 (5.5%)	0.130
Haematological or Coagulation Disorders	289 (20.2%)	289 (9.2%)	<0.001
Chronic treatment with steroids or NSAIDS	142 (9.9%)	299 (9.5%)	0.627
Clinical indication for 12 months OAC	481 (33.7%)	1185 (37.6%)	0.011
PRECISE DAPT score¶	34.24 ± 10.13	23.37 ± 9.59	<0.001
Prior bleeding	85 (6.0%)	235 (7.5%)	0.070
Hemoglobin, g/L	12.62 ± 1.85	13.49 ± 1.69	<0.001
WBC¶, 10^9/L	8.44 ± 4.12	8.05 ± 9.80	0.147
CrCl (MDRD), ml/min/1.73 m²	47.16 ± 16.14	81.60 ± 18.76	<0.001
Indication*			
Stable angina	518 (36.3%)	1331 (42.2%)	<0.001
Silent ischemia	147 (10.3%)	372 (11.8%)	0.145
NSTEMI	436 (30.5%)	717 (22.8%)	<0.001
STEMI	176 (12.3%)	362 (11.5%)	0.428
Unstable angina	151 (10.6%)	369 (11.7%)	0.269

Abbreviations: BMI, body mass index; CABG, coronary artery bypass grafting; CAD, coronary artery disease; CKD, chronic kidney disease; CrCl, creatinine clearance; HF, heart failure; LVEF, left ventricular ejection fraction; MI, myocardial infarction; OAC, oral anticoagulation; PCI, percutaneous coronary intervention; WBC, white blood cell.

Data are mean (standard deviation), n (%), or n/n (%) in case of missing data.

*Data from first PCI only.

¶calculated at screening visit. n=1 PRECISE Score calculated without risk due to WBC.

Supplementary Table 3. Procedural characteristics among CKD and non-CKD patients.

	CKD (n=1428)	Non-CKD (n=3151)	p-value
Arterial access site			
Femoral access	251 (17.6%)	402 (12.8%)	<0.001
Radial access	1172 (82.1%)	2742 (87.0%)	<0.001
Brachial access	5 (0.4%)	7 (0.2%)	0.533
Total amount of contrast, cc	165.96 ± 81.32	168.18 ± 79.27	0.385
Medications during the procedure*			
Unfractionated heparin	1367 (95.7%)	2989 (94.9%)	0.235
Bivalirudin	5 (0.4%)	2 (0.1%)	0.034
Low molecular weight heparin	31 (2.2%)	96 (3.0%)	0.099
Cangrelor	3 (0.2%)	8 (0.3%)	1.000
Glycoprotein II/IIIa inhibitors	51 (3.6%)	111 (3.5%)	0.931
Total number of PCIs¶			
one	1295 (90.7%)	2864 (90.9%)	0.825
two	130 (9.1%)	275 (8.7%)	0.694
three	3 (0.2%)	12 (0.4%)	0.417
Total nr of vessels treated per patient¥			
one	1009 (70.7%)	2356 (74.8%)	0.004
two	361 (25.3%)	663 (21.0%)	0.002
three	58 (4.1%)	132 (4.2%)	0.873
Treated vessel(s) per patient			
Left main	102 (7.1%)	158 (5.0%)	0.005
Left arterial descending artery	748 (52.4%)	1763 (56.0%)	0.025
Left circumflex artery	446 (31.2%)	895 (28.4%)	0.054
Right coronary artery	525 (36.8%)	1135 (36.0%)	0.642
Bypass graft	28 (2.0%)	48 (1.5%)	0.318
Total nr of treated lesions per patient			
one	943 (66.0%)	2172 (68.9%)	0.055
two	354 (24.8%)	671 (21.3%)	0.009
three or more	131 (9.2%)	308 (9.8%)	0.551
At least one complex lesion B2 or C	1048 (73.4%)	2093 (66.4%)	<0.001
Any Bifurcation or trifurcation stenting§	65 (4.6%)	119 (3.8%)	0.223
Any OCT of treated lesion	31 (2.2%)	103 (3.3%)	0.046
Any IVUS of treated lesion	77 (5.4%)	210 (6.7%)	0.1

Treated lesions	n = 2072	n = 4562	
Lesion location			
Left main	104 (5.0%)	159 (3.5%)	0.003
Left arterial descending artery	823 (39.7%)	2003 (43.9%)	0.001
Left circumflex artery	502 (24.2%)	993 (21.8%)	0.023
Right coronary artery	612 (29.5%)	1355 (29.7%)	0.896
Bypass graft	28 (1.4%)	44 (1.0%)	0.223
SVG	4 (0.2%)	11 (0.2%)	0.703
LIMA/RIMA/Radial graft	312 (15.1%)	744 (16.3%)	0.213
Bifurcation or trifurcation disease per lesion	47 (2.3%)	104 (2.3%)	0.979
Rotablator	104 (5.0%)	159 (3.5%)	0.003

Abbreviations: APT, antiplatelet therapy; CKD, chronic kidney disease; DAPT, dual antiplatelet therapy; IABP, intra-aortic balloon pump; IVUS, intravascular ultrasound; LIMA, left internal mammary artery; LVAD, left ventricular assist device; RIMA, right internal mammary artery; SVG, saphenous vein graft; TIMI: Thrombolysis in Myocardial Infarction, OCT, optical coherence tomography; PCI, percutaneous coronary intervention.
Data are mean (SD), n (%), or n/n (%) in case of missing data.

* Data from first PCI only.
¶ One PCI and up to two staged PCIs. The last PCI was the qualifying PCI 1 month before the randomization.
§ into both main- and side-branch.
¥ Left main counted as two vessels.

Supplementary Table 4. Baseline characteristics by randomised antiplatelet regimen and CKD status.

	CKD			Non-CKD		
	Abbreviated DAPT (n=711)	**Standard DAPT(n=717)**	**p-value**	**Abbreviated DAPT (n=1584)**	**Standard DAPT (n=1567)**	**p-value**
Age, years	77.30 ± 7.99	77.41 ± 8.19	0.799	75.58 ± 8.96	75.29 ± 8.95	0.367
Male sex	462 (65.0%)	442 (61.6%)	0.207	1128 (71.2%)	1139 (72.7%)	0.362
BMI	27.62 ± 4.78	27.85 ± 4.83	0.377	27.09 ± 4.63	27.26 ± 4.69	0.313
Family history of CAD	159 (22.4%)	161 (22.5%)	1.000	397 (25.1%)	392 (25.0%)	1.000
Arterial hypertension	602 (84.7%)	604 (84.2%)	0.827	1164 (73.5%)	1183 (75.5%)	0.205
Hyperlipidemia	484 (68.1%)	477 (66.5%)	0.536	1058 (66.8%)	1078 (68.8%)	0.237
Current smoking	58 (8.2%)	55 (7.7%)	0.769	172 (10.9%)	129 (8.3%)	0.013
LVEF	51.24 ± 12.09	51.24 ± 12.19	0.996	54.50 ± 10.99	53.76 ± 11.49	0.078
Prior MI	165 (23.2%)	153 (21.3%)	0.409	269 (17.0%)	277 (17.7%)	0.638
Prior PCI	206 (29.0%)	195 (27.2%)	0.480	388 (24.5%)	399 (25.5%)	0.537
Prior cerebrovascular event	94 (13.2%)	93 (13.0%)	0.938	174 (11.0%)	209 (13.3%)	0.044
Prior CABG	62 (8.7%)	67 (9.3%)	0.712	108 (6.8%)	104 (6.6%)	0.887
Diabetes mellitus	265 (37.3%)	290 (40.4%)	0.232	489 (30.9%)	494 (31.5%)	0.701
Active cancer	30 (4.2%)	33 (4.6%)	0.797	80 (5.1%)	93 (5.9%)	0.309
Haematological or Coagulation Disorders	141 (19.8%)	148 (20.6%)	0.742	149 (9.4%)	140 (8.9%)	0.666
Chronic treatment with steroids or NSAIDS	61 (8.6%)	81 (11.3%)	0.093	141 (8.9%)	158 (10.1%)	0.274
Clinical indication for 12 months OAC	252 (35.4%)	229 (31.9%)	0.162	596 (37.6%)	589 (37.6%)	1.000
PRECISE DAPT score¶	33.87 ± 10.09	34.61 ± 10.16	0.168	23.64 ± 9.72	23.10 ± 9.45	0.109
Prior bleeding	42 (5.9%)	43 (6.0%)	1.000	123 (7.8%)	112 (7.1%)	0.542
Hemoglobin, g/L	12.70 ± 1.84	12.53 ± 1.86	0.086	13.47 ± 1.70	13.50 ± 1.68	0.669
WBC¶, 10^9/L	8.33 ± 3.93	8.55 ± 4.30	0.299	8.27 ± 13.51	7.83 ± 2.87	0.204

CrCl (MDRD), ml/min/1.73 m²	47.10 ± 15.98	47.23 ± 16.32	0.874	81.32 ± 18.87	81.88 ± 18.65	0.405
Indication*						
Stable angina	257 (36.1%)	261 (36.4%)	0.956	665 (42.0%)	666 (42.5%)	0.773
Silent ischemia	72 (10.1%)	75 (10.5%)	0.862	173 (10.9%)	199 (12.7%)	0.123
NSTEMI	216 (30.4%)	220 (30.7%)	0.909	379 (23.9%)	338 (21.6%)	0.116
STEMI	96 (13.5%)	80 (11.2%)	0.198	177 (11.2%)	185 (11.8%)	0.615
Unstable angina	70 (9.8%)	81 (11.3%)	0.390	190 (12.0%)	179 (11.4%)	0.619

Abbreviations: BMI, body mass index; CABG, coronary artery bypass grafting; CAD, coronary artery disease; CKD, chronic kidney disease; CrCl, creatinine clearance; DAPT, dual antiplatelet therapy; HF, heart failure; LVEF, left ventricular ejection fraction; MI, myocardial infarction; NSAIDs, non-steroidal anti-inflammatory drugs; OAC, oral anticoagulation; PCI, percutaneous coronary intervention; WBC, white blood cell.

Data are mean (SD), n (%), or n/n (%) in case of missing data.

* Data from first PCI only.

¶ calculated at screening visit; n=1 PRECISE Score calculated without risk caused by white blood cell.

Supplementary Table 5. Procedural characteristics by randomised antiplatelet regimen and CKD status.

	CKD			Non-CKD		
	Abbreviated DAPT (n=711)	**Standard DAPT(n=717)**	**p-value**	**Abbreviated DAPT (n=1584)**	**Standard DAPT (n=1567)**	**p-value**
Arterial access site						
Femoral access	132 (18.6%)	119 (16.6%)	0.331	228 (14.4%)	174 (11.1%)	0.006
Radial access	576 (81.0%)	596 (83.1%)	0.302	1354 (85.5%)	1388 (88.6%)	0.011
Brachial access	3 (0.4%)	2 (0.3%)	0.686	2 (0.1%)	5 (0.3%)	0.286
Total amount of contrast, cc	168.07 ± 85.00	163.87 ± 77.50	0.332	168.21 ± 78.30	168.16 ± 80.26	0.985
Medications during the procedure*						
Unfractionated heparin	680 (95.6%)	687 (95.8%)	0.896	1504 (94.9%)	1485 (94.8%)	0.936
Bivalirudin	4 (0.6%)	1 (0.1%)	0.216	1 (0.1%)	1 (0.1%)	1.000
Low molecular weight heparin	17 (2.4%)	14 (2.0%)	0.591	46 (2.9%)	50 (3.2%)	0.679
Cangrelor	3 (0.4%)	0 (0.0%)	0.123	5 (0.3%)	3 (0.2%)	0.726
Glycoprotein II/IIIa inhibitors	27 (3.8%)	24 (3.3%)	0.671	59 (3.7%)	52 (3.3%)	0.563
Total number of PCIs¶						
one	641 (90.2%)	654 (91.2%)	0.524	1452 (91.7%)	1412 (90.1%)	0.137
two	69 (9.7%)	61 (8.5%)	0.462	122 (7.7%)	153 (9.8%)	0.043
three	1 (0.1%)	2 (0.3%)	1.000	10 (0.6%)	2 (0.1%)	0.038
Total nr of vessels treated per patient¥						
one	501 (70.5%)	508 (70.9%)	0.907	1215 (76.7%)	1141 (72.8%)	0.012
two	180 (25.3%)	181 (25.2%)	1.000	303 (19.1%)	360 (23.0%)	0.009
three	30 (4.2%)	28 (3.9%)	0.790	66 (4.2%)	66 (4.2%)	1.000
Treated vessel(s) per patient						
Left main	49 (6.9%)	53 (7.4%)	0.758	77 (4.9%)	81 (5.2%)	0.744
Left arterial descending artery	370 (52.0%)	378 (52.7%)	0.832	870 (54.9%)	893 (57.0%)	0.251
Left circumflex artery	225 (31.6%)	221 (30.8%)	0.775	427 (27.0%)	468 (29.9%)	0.075

Right coronary artery	270 (38.0%)	255 (35.6%)	0.351	584 (36.9%)	551 (35.2%)	0.335
Bypass graft	14 (2.0%)	14 (2.0%)	1.000	24 (1.5%)	24 (1.5%)	1.000
Total nr of treated lesions per patient						
one	468 (65.8%)	475 (66.2%)	0.867	1111 (70.1%)	1061 (67.7%)	0.144
two	177 (24.9%)	177 (24.7%)	0.951	326 (20.6%)	345 (22.0%)	0.338
three or more	66 (9.3%)	65 (9.1%)	0.927	147 (9.3%)	161 (10.3%)	0.368
At least one complex lesion B2 or C	507 (71.3%)	541 (75.5%)	0.082	1055 (66.6%)	1038 (66.2%)	0.850
Any Bifurcation or trifurcation stenting§	33 (4.6%)	32 (4.5%)	0.900	50 (3.2%)	69 (4.4%)	0.076
Any OCT of treated lesion	11 (1.5%)	20 (2.8%)	0.145	51 (3.2%)	52 (3.3%)	0.920
Any IVUS of treated lesion	35 (4.9%)	42 (5.9%)	0.483	100 (6.3%)	110 (7.0%)	0.433
Treated lesions	n = 1034	n = 1038		n = 2260	n = 2302	
Lesion location						
Left main	50 (4.8%)	54 (5.2%)	0.701	78 (3.5%)	81 (3.5%)	0.900
Left arterial descending artery	411 (39.7%)	412 (39.7%)	0.978	983 (43.5%)	1020 (44.3%)	0.577
Left circumflex artery	251 (24.3%)	251 (24.2%)	0.959	476 (21.1%)	517 (22.5%)	0.238
Right coronary artery	306 (29.6%)	306 (29.5%)	0.957	699 (30.9%)	656 (28.5%)	0.084
Bypass graft						
SVG	14 (1.4%)	14 (1.3%)	0.993	20 (0.9%)	24 (1.0%)	0.630
LIMA/RIMA/Radial graft	2 (0.2%)	2 (0.2%)	0.997	7 (0.3%)	4 (0.2%)	0.356
Bifurcation or trifurcation disease per lesion	153 (14.8%)	159 (15.3%)	0.751	382 (16.9%)	362 (15.7%)	0.291
Rotablator	22 (2.1%)	25 (2.4%)	0.692	56 (2.5%)	48 (2.1%)	0.410

Abbreviations: CKD, chronic kidney disease; DAPT, antiplatelet therapy; IABP, intra-aortic balloon pump; IVUS, intravascular ultrasound; LVAD, left ventricular assist device; OCT, optical coherence tomography; PCI, percutaneous coronary intervention.

Data are mean (SD), n (%), or n/n (%) in case of missing data.
* Data from first PCI only.

¶ One PCI and up to two staged PCIs. The last PCI was the qualifying PCI 1 month before the randomization.
§ into both main- and side-branch.
¥ Left main counted as two vessels.

Supplementary Table 6. Clinical outcomes at 11 months after randomisation in CKD and non-CKD patients randomised to abbreviated versus standard DAPT.

	CKD					Non-CKD					
	Abbreviated DAPT (n=711)	**Standard DAPT (n=717)**	**Hazard ratio (95% CI)**	**p-value**	**Com-Nogue Risk Difference [95% CI]**	**Abbreviated DAPT (n=1584)**	**Standard DAPT (n=1567)**	**Hazard ratio (95% CI)**	**p-value**	**Com-Nogue Risk Difference (95% CI)**	**interaction p-value**
NACE	75 (10.60)	83 (11.59)	0.91 (0.66-1.24)	0.540	-1.00 [-4.26 to 2.27]	97 (6.15)	99 (6.35)	0.96 (0.73-1.27)	0.792	-0.21 [-1.90 to 1.49]	0.780
MACCE	63 (8.90)	70 (9.78)	0.91 (0.64-1.27)	0.570	-0.88 [-3.90 to 2.15]	75 (4.76)	68 (4.37)	1.09 (0.78-1.51)	0.610	0.39 [-1.07 to 1.85]	0.445
MCB	51 (7.33)	68 (9.63)	0.74 (0.52-1.07)	0.106	-2.30 [-5.21 to 0.61]	97 (6.19)	143 (9.23)	0.66 (0.51-0.85)	0.001	-3.04 [-4.92 to -1.17]	0.590
Death	39 (5.52)	46 (6.43)	0.85 (0.56-1.31)	0.463	-0.91 [-3.37 to 1.55]	36 (2.28)	35 (2.25)	1.01 (0.64-1.62)	0.952	0.03 [-1.01 to 1.08]	0.589
Cardiovascular Death	19 (2.73)	24 (3.40)	0.80 (0.44-1.45)	0.458	-0.67 [-2.47 to 1.13]	18 (1.15)	20 (1.29)	0.89 (0.47-1.68)	0.714	-0.14 [-0.92 to 0.63]	0.808
Non-cardiovascular Death	14 (2.02)	15 (2.15)	0.94 (0.45-1.94)	0.864	-0.13 [-1.63 to 1.37]	15 (0.96)	13 (0.84)	1.14 (0.54-2.39)	0.733	0.12 [-0.54 to 0.78]	0.717
Undetermined Death	6 (0.87)	7 (1.00)	0.86 (0.29-2.56)	0.787	-0.14 [-1.15 to 0.87]	3 (0.19)	2 (0.13)	1.48 (0.25-8.86)	0.668	0.06 [-0.22 to 0.34]	0.611
Cerebrovascular accident	8 (1.15)	15 (2.15)	0.54 (0.23-1.26)	0.153	-1.00 [-2.34 to 0.33]	9 (0.58)	17 (1.10)	0.52 (0.23-1.17)	0.114	-0.52 [-1.17 to 0.12]	0.965
Stroke ¶	5 (0.71)	11 (1.58)	0.46 (0.16-1.32)	0.147	-0.87 [-1.98 to 0.25]	7 (0.45)	12 (0.78)	0.58 (0.23-1.46)	0.245	-0.33 [-0.88 to 0.22]	0.749
ischemic Stroke	4 (0.56)	9 (1.29)	0.45 (0.14-1.45)	0.181	-0.73 [-1.73 to 0.28]	7 (0.45)	9 (0.58)	0.77 (0.29-2.06)	0.599	-0.14 [-0.64 to 0.37]	0.491
hemorrhagic Stroke	1 (0.15)	3 (0.44)	0.33 (0.03-3.21)	0.343	-0.29 [-0.86 to 0.29]	0 (0.00)	2 (0.13)	0.20 (0.01-4.16)	0.247	-0.13 [-0.31 to 0.05]	-
TIA	3 (0.44)	4 (0.57)	0.75 (0.17-3.37)	0.711	-0.14 [-0.89 to 0.61]	2 (0.13)	5 (0.32)	0.39 (0.08-2.03)	0.266	-0.19 [-0.53 to 0.14]	0.567
Myocardial infarction	27 (3.90)	22 (3.14)	1.24 (0.70-2.17)	0.458	0.76 [-1.18 to 2.69]	33 (2.12)	27 (1.75)	1.21 (0.73-2.01)	0.466	0.36 [-0.61 to 1.33]	0.950
Definite or Probable ST	6 (0.87)	3 (0.43)	2.01 (0.50-8.06)	0.322	0.45 [-0.40 to 1.29]	8 (0.51)	6 (0.39)	1.32 (0.46-3.80)	0.610	0.12 [-0.35 to 0.59]	0.632

Bleeding (BARC classification)											
BARC type 1	17 (2.44)	34 (4.83)	0.50 (0.28-0.89)	0.018	-2.39 [-4.35 to -0.44]	48 (3.06)	75 (4.84)	0.63 (0.44-0.90)	0.011	-1.78 [-3.15 to -0.41]	0.501
BARC type 2	30 (4.31)	44 (6.26)	0.68 (0.43-1.08)	0.100	-1.95 [-4.30 to 0.39]	72 (4.60)	108 (6.98)	0.65 (0.48-0.87)	0.004	-2.38 [-4.02 to -0.74]	0.880
BARC type 3	23 (3.32)	21 (2.99)	1.10 (0.61-1.99)	0.752	0.33 [-1.50 to 2.17]	30 (1.92)	38 (2.46)	0.78 (0.48-1.25)	0.298	-0.54 [-1.57 to 0.49]	0.368
BARC type 3a	11 (1.59)	15 (2.14)	0.73 (0.34-1.60)	0.437	-0.55 [-1.97 to 0.86]	15 (0.96)	15 (0.97)	0.99 (0.48-2.02)	0.968	-0.01 [-0.69 to 0.68]	0.586
BARC type 3b	10 (1.46)	4 (0.56)	2.52 (0.79-8.03)	0.118	0.89 [-0.16 to 1.94]	11 (0.70)	16 (1.04)	0.68 (0.31-1.46)	0.320	-0.33 [-0.99 to 0.32]	0.064
BARC type 3c	3 (0.43)	2 (0.29)	1.51 (0.25-9.03)	0.653	0.15 [-0.48 to 0.78]	4 (0.26)	7 (0.45)	0.56 (0.16-1.92)	0.360	-0.20 [-0.61 to 0.22]	0.373
BARC type 4	0 (0.00)	0 (0.00)	-	-	-	0 (0.00)	0 (0.00)	-	-	-	-
BARC type 5	2 (0.30)	4 (0.57)	0.50 (0.09-2.75)	0.427	-0.28 [-0.97 to 0.42]	0 (0.00)	4 (0.26)	0.11 (0.01-2.04)	0.061	-0.26 [-0.52 to -0.01]	-
BARC type 5a	0 (0.00)	1 (0.14)	0.34 (0.01-8.33)	1.000	-0.14 [-0.41 to 0.13]	0 (0.00)	1 (0.07)	0.33 (0.01-8.09)	0.497	-0.07 [-0.19 to 0.06]	-
BARC type 5b	2 (0.30)	3 (0.43)	0.67 (0.11-4.01)	0.661	-0.14 [-0.78 to 0.50]	0 (0.00)	3 (0.19)	0.14 (0.01-2.71)	0.123	-0.19 [-0.41 to 0.03]	-
BARC type 3 or 5	25 (3.61)	25 (3.55)	1.00 (0.58-1.75)	0.988	0.06 [-1.89 to 2.01]	30 (1.92)	42 (2.71)	0.70 (0.44-1.12)	0.138	-0.80 [-1.85 to 0.26]	0.333

Abbreviations: BARC, Bleeding Academic Research Consortium; CI, confidence interval; CKD, chronic kidney disease; HR, hazard ratio; MACCE, major adverse cardiac and cerebral events; MCB, major or clinically relevant non-major bleeding; NACE, net adverse clinical events; ST, stent thrombosis; TIA, transient ischemic attack.

Nr of first events of each type (Kaplan-Meier failure %). Hazard ratio (95% CI) from Cox's time-to-first event analyses in ITT population. Continuity corrected risk ratios (95% CI) in case of zero events with Fisher's exact test p-value. Interaction p-value testing for modifying effect of CKD (yes or no) on the hazard ratio scale.

¶includes undetermined Strokes.

Supplementary Table 7. Clinical outcomes at 11 months after randomisation (12-month follow-up) in CKD and non-CKD patients with a clinical indication for 12-month OAC.

	CKD					Non-CKD					
	Abbreviated DAPT (N=252)	**Standard DAPT (N=229)**	**Hazard ratio (95% CI)**	**p-value**	**Com-Nogue Risk Difference (95% CI)**	**Abbreviated DAPT (N=596)**	**Standard DAPT (N=589)**	**Hazard ratio (95% CI)**	**p-value**	**Com-Nogue Risk Difference (95% CI)**	**interaction p-value**
NACE	34 (13.49)	33 (14.46)	0.93 (0.57-1.49)	0.753	-0.97 [-7.18 to 5.25]	34 (5.73)	45 (7.72)	0.73 (0.47-1.14)	0.172	-1.99 [-4.85 to 0.87]	0.483
MACCE	28 (11.11)	27 (11.83)	0.93 (0.55-1.58)	0.789	-0.72 [-6.43 to 4.99]	22 (3.71)	27 (4.64)	0.80 (0.45-1.40)	0.430	-0.93 [-3.22 to 1.36]	0.693
MCB	30 (12.12)	32 (14.24)	0.83 (0.50-1.36)	0.457	-2.12 [-8.24 to 4.00]	53 (8.97)	62 (10.65)	0.83 (0.57-1.19)	0.310	-1.68 [-5.08 to 1.73]	0.994
Death	18 (7.16)	20 (8.77)	0.81 (0.43-1.53)	0.513	-1.61 [-6.47 to 3.25]	13 (2.19)	13 (2.24)	0.98 (0.46-2.12)	0.965	-0.04 [-1.73 to 1.64]	0.702
Cardiovascular death	10 (4.06)	10 (4.47)	0.90 (0.37-2.16)	0.813	-0.40 [-4.06 to 3.26]	6 (1.01)	11 (1.90)	0.54 (0.20-1.45)	0.219	-0.88 [-2.25 to 0.49]	0.444
Non-cardiovascular death	5 (2.04)	5 (2.30)	0.90 (0.26-3.10)	0.863	-0.25 [-2.92 to 2.41]	6 (1.02)	2 (0.35)	2.95 (0.60-14.62)	0.185	0.67 [-0.27 to 1.61]	0.250
Undetermined death	3 (1.21)	5 (2.26)	0.54 (0.13-2.26)	0.397	-1.04 [-3.43 to 1.34]	1 (0.17)	0 (0.00)	2.96 (0.12-72.51)	1.000	0.17 [-0.17 to 0.51]	-
Cerebrovascular accident	1 (0.40)	6 (2.74)	0.15 (0.02-1.24)	0.078	-2.34 [-4.64 to -0.04]	2 (0.34)	7 (1.21)	0.28 (0.06-1.35)	0.112	-0.86 [-1.87 to 0.14]	0.641
Stroke¶	1 (0.40)	5 (2.27)	0.18 (0.02-1.54)	0.117	-1.87 [-3.99 to 0.24]	1 (0.17)	5 (0.86)	0.20 (0.02-1.68)	0.138	-0.69 [-1.51 to 0.13]	0.954
TIA	0 (0.00)	1 (0.47)	0.30 (0.01-7.33)	0.476	-0.47 [-1.39 to 0.45]	1 (0.17)	2 (0.35)	0.49 (0.04-5.41)	0.561	-0.17 [-0.76 to 0.41]	-
Myocardial infarction	10 (4.14)	6 (2.72)	1.51 (0.55-4.16)	0.423	1.41 [-1.89 to 4.72]	9 (1.53)	11 (1.90)	0.80 (0.33-1.94)	0.628	-0.37 [-1.86 to 1.12]	0.357
Definite or Probable ST	2 (0.84)	1 (0.46)	1.81 (0.16-19.92)	0.629	0.38 [-1.09 to 1.84]	1 (0.17)	3 (0.52)	0.33 (0.03-3.14)	0.333	-0.35 [-1.03 to 0.32]	0.310
Bleeding (BARC classification)											
Type 1	7 (2.85)	12 (5.37)	0.52 (0.20-1.31)	0.164	-2.52 [-6.14 to 1.09]	27 (4.58)	35 (6.01)	0.75 (0.46-1.24)	0.265	-1.43 [-4.00 to 1.13]	0.486

Type 2	19 (7.68)	22 (9.85)	0.77 (0.41-1.42)	0.396	-2.17 [-7.30 to 2.96]	41 (6.95)	43 (7.39)	0.93 (0.61-1.43)	0.739	-0.44 [-3.40 to 2.51]	0.611
Type 3	12 (4.90)	10 (4.49)	1.08 (0.47-2.51)	0.851	0.40 [-3.44 to 4.24]	14 (2.37)	23 (3.96)	0.59 (0.31-1.15)	0.124	-1.58 [-3.59 to 0.42]	0.272
Type 3a	5 (2.03)	7 (3.15)	0.64 (0.20-2.02)	0.448	-1.12 [-4.02 to 1.78]	6 (1.02)	11 (1.89)	0.54 (0.20-1.45)	0.218	-0.88 [-2.25 to 0.50]	0.814
Type 3b	7 (2.88)	1 (0.45)	6.36 (0.78-51.68)	0.084	2.43 [0.16 to 4.71]	6 (1.02)	11 (1.89)	0.53 (0.20-1.45)	0.217	-0.88 [-2.25 to 0.50]	0.037
Type 3c	1 (0.42)	2 (0.91)	0.45 (0.04-4.96)	0.515	-0.49 [-1.99 to 1.01]	2 (0.34)	1 (0.17)	1.97 (0.18-21.70)	0.581	0.17 [-0.41 to 0.75]	0.395
Type 4	0 (0.00)	0 (0.00)	-	-	-	0 (0.00)	0 (0.00)	-	-	-	-
Type 5	1 (0.42)	1 (0.46)	0.90 (0.06-14.41)	0.941	-0.04 [-1.26 to 1.18]	0 (0.00)	2 (0.35)	0.20 (0.01-4.16)	0.247	-0.35 [-0.83 to 0.13]	-
Type 5a	0 (0.00)	0 (0.00)	-	-	-	0 (0.00)	1 (0.18)	0.33 (0.01-8.08)	0.497	-0.18 [-0.52 to 0.17]	1.000
Type 5b	1 (0.42)	1 (0.46)	0.90 (0.06-14.41)	0.941	-0.04 [-1.26 to 1.18]	0 (0.00)	1 (0.17)	0.33 (0.01-8.08)	0.497	-0.17 [-0.51 to 0.17]	-
Type 3 or 5	13 (5.31)	11 (4.94)	1.07 (0.48-2.39)	0.871	0.37 [-3.63 to 4.37]	14 (2.37)	25 (4.30)	0.55 (0.28-1.05)	0.070	-1.93 [-3.99 to 0.13]	0.206

Abbreviations: BARC, Bleeding Academic Research Consortium; CI, confidence interval; CKD, chronic kidney disease; MACCE, major adverse cardiac and cerebral events; MCB, major or clinically relevant non-major bleeding; NACE, net adverse clinical events; ST, stent thrombosis; TIA, transient ischemic attack.

Nr of first events of each type (Kaplan-Meier failure %). Hazard ratio (95% CI) from Cox's time-to-first event analyses in ITT population. Continuity corrected risk ratios (95% CI) in case of zero events with Fisher's exact test p-value. Interaction p-value testing for modifying effect of CKD (yes or no) on the hazard ratio scale.
¶includes undetermined Strokes.

Supplementary Table 8. Clinical outcomes at 11 months after randomisation (12-month follow-up) in CKD and non-CKD patients without a clinical indication for 12-month OAC.

	CKD					Non-CKD					
	Abbreviated DAPT (n=459)	**Standard DAPT (n=488)**	**Hazard ratio (95% CI)**	**p-value**	**Com-Nogue Risk Difference (95% CI)**	**Abbreviated DAPT (n=988)**	**Standard DAPT (n=978)**	**Hazard ratio (95% CI)**	**p-value**	**Com-Nogue Risk Difference (95% CI)**	**interaction p-value**
NACE	41 (8.99)	50 (10.25)	0.87 (0.58-1.31)	0.509	-1.26 [-5.02 to 2.50]	63 (6.40)	54 (5.54)	1.16 (0.80-1.66)	0.434	0.86 [-1.23 to 2.96]	0.311
MACCE	35 (7.68)	43 (8.82)	0.87 (0.56-1.36)	0.534	-1.14 [-4.65 to 2.37]	53 (5.39)	41 (4.21)	1.28 (0.85-1.93)	0.229	1.18 [-0.71 to 3.07]	0.204
MCB	21 (4.69)	36 (7.47)	0.61 (0.36-1.04)	0.072	-2.78 [-5.84 to 0.28]	44 (4.51)	81 (8.39)	0.52 (0.36-0.76)	0.001	-3.88 [-6.06 to -1.70]	0.653
Death	21 (4.61)	26 (5.33)	0.86 (0.48-1.53)	0.603	-0.72 [-3.49 to 2.05]	23 (2.34)	22 (2.26)	1.03 (0.58-1.85)	0.913	0.08 [-1.24 to 1.41]	0.659
Cardiovascular death	9 (1.99)	14 (2.90)	0.68 (0.30-1.58)	0.373	-0.91 [-2.89 to 1.07]	12 (1.23)	9 (0.93)	1.32 (0.56-3.13)	0.531	0.30 [-0.62 to 1.22]	0.285
Non-cardiovascular death	9 (2.01)	10 (2.09)	0.96 (0.39-2.36)	0.924	-0.08 [-1.91 to 1.74]	9 (0.92)	11 (1.13)	0.81 (0.33-1.95)	0.636	-0.21 [-1.11 to 0.68]	0.792
Undetermined death	3 (0.67)	2 (0.42)	1.59 (0.27-9.54)	0.610	0.25 [-0.70 to 1.21]	2 (0.20)	2 (0.21)	0.99 (0.14-7.02)	0.991	0.00 [-0.41 to 0.40]	0.724
Cerebrovascular accident	7 (1.56)	9 (1.89)	0.83 (0.31-2.22)	0.706	-0.33 [-2.00 to 1.35]	7 (0.72)	10 (1.04)	0.69 (0.26-1.82)	0.453	-0.32 [-1.15 to 0.52]	0.799
Stroke¶	4 (0.88)	6 (1.26)	0.71 (0.20-2.52)	0.597	-0.38 [-1.70 to 0.95]	6 (0.62)	7 (0.73)	0.85 (0.28-2.52)	0.766	-0.11 [-0.84 to 0.62]	0.836
TIA	3 (0.67)	3 (0.63)	1.06 (0.21-5.26)	0.942	0.05 [-0.99 to 1.09]	1 (0.10)	3 (0.31)	0.33 (0.03-3.17)	0.336	-0.21 [-0.61 to 0.20]	0.408
Myocardial infarction	17 (3.78)	16 (3.34)	1.13 (0.57-2.24)	0.724	0.44 [-1.94 to 2.82]	24 (2.47)	16 (1.66)	1.49 (0.79-2.80)	0.217	0.80 [-0.46 to 2.07]	0.562
Definite or Probable ST	4 (0.90)	2 (0.41)	2.13 (0.39-11.60)	0.384	0.48 [-0.56 to 1.53]	7 (0.72)	3 (0.31)	2.31 (0.60-8.95)	0.224	0.41 [-0.23 to 1.04]	0.940
Bleeding (BARC classification)											
Type 1	10 (2.21)	22 (4.58)	0.48 (0.23-1.01)	0.052	-2.36 [-4.67 to -0.05]	21 (2.15)	40 (4.14)	0.52 (0.30-0.87)	0.014	-1.99 [-3.54 to -0.44]	0.867

Type 2	11 (2.46)	22 (4.59)	0.53 (0.25-1.08)	0.081	-2.14 [-4.50 to 0.22]	31 (3.18)	65 (6.74)	0.46 (0.30-0.71)	<0.001	-3.55 [-5.48 to -1.63]	0.768
Type 3	11 (2.46)	11 (2.29)	1.06 (0.46-2.44)	0.891	0.17 [-1.79 to 2.13]	16 (1.64)	15 (1.56)	1.05 (0.52-2.13)	0.888	0.09 [-1.03 to 1.20]	0.990
Type 3a	6 (1.34)	8 (1.67)	0.79 (0.28-2.29)	0.670	-0.33 [-1.90 to 1.24]	9 (0.93)	4 (0.41)	2.22 (0.68-7.22)	0.183	0.51 [-0.21 to 1.24]	0.202
Type 3b	3 (0.68)	3 (0.62)	1.06 (0.21-5.26)	0.942	0.06 [-0.98 to 1.09]	5 (0.51)	5 (0.52)	0.99 (0.29-3.41)	0.985	-0.01 [-0.65 to 0.63]	0.945
Type 3c	2 (0.44)	0 (0.00)	5.32 (0.26-110.52)	0.235	0.44 [-0.17 to 1.05]	2 (0.21)	6 (0.62)	0.33 (0.07-1.63)	0.173	-0.42 [-0.99 to 0.16]	-
Type 4	0 (0.00)	0 (0.00)	-	-	-	0 (0.00)	0 (0.00)	-	-	-	-
Type 5	1 (0.23)	3 (0.63)	0.35 (0.04-3.41)	0.369	-0.40 [-1.23 to 0.44]	0 (0.00)	2 (0.21)	0.20 (0.01-4.16)	0.247	-0.21 [-0.50 to 0.08]	-
Type 5a	0 (0.00)	1 (0.20)	0.35 (0.01-8.57)	1.000	-0.20 [-0.61 to 0.20]	0 (0.00)	0 (0.00)	-	-	-	1.000
Type 5b	1 (0.23)	2 (0.42)	0.53 (0.05-5.86)	0.606	-0.19 [-0.93 to 0.54]	0 (0.00)	2 (0.21)	0.20 (0.01-4.16)	0.247	-0.21 [-0.50 to 0.08]	-
Type 3 or 5	12 (2.68)	14 (2.91)	0.91 (0.42-1.96)	0.808	-0.22 [-2.34 to 1.90]	16 (1.64)	17 (1.76)	0.93 (0.47-1.84)	0.830	-0.12 [-1.27 to 1.03]	0.967

Abbreviations: BARC, Bleeding Academic Research Consortium; CI, confidence interval; CKD, chronic kidney disease; MACCE, major adverse cardiac and cerebral events; MCB, major or clinically relevant non-major bleeding; NACE, net adverse clinical events; ST, stent thrombosis; TIA, transient ischemic attack.

Nr of first events of each type (Kaplan-Meier failure %). Hazard ratio (95% CI) from Cox's time-to-first event analyses in ITT population. Continuity corrected risk ratios (95% CI) in case of zero events with Fisher's exact test p-value. Interaction p-value testing for modifying effect of CKD (yes or no) on the hazard ratio scale.
¶includes undetermined Strokes.

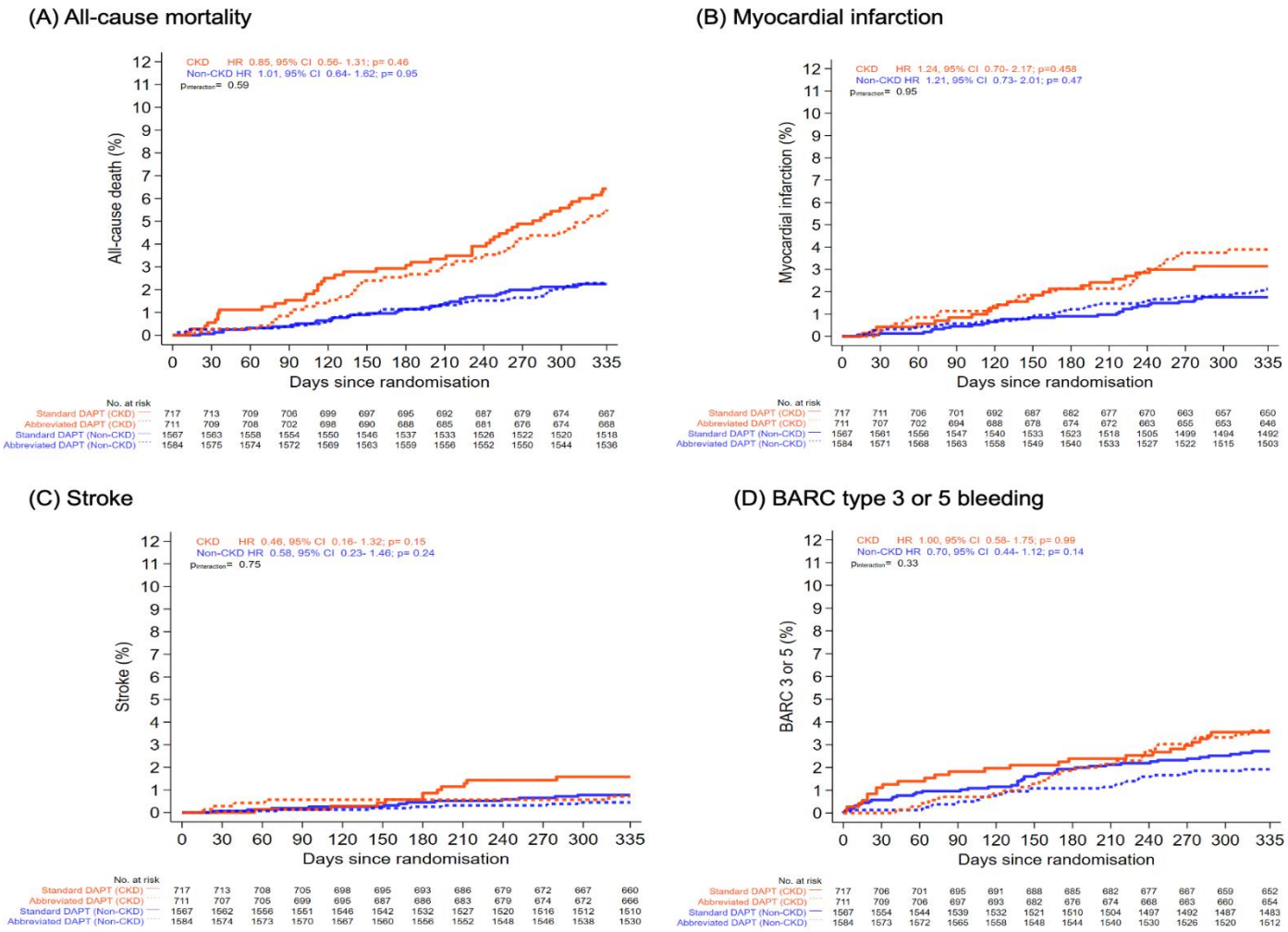
Supplementary Table 9. Clinical endpoints at 11 months after randomisation (12-month follow-up) according to renal dysfunction severity (eGFR thresholds at 45 and 60 mL/min/1.73 m^2).

	eGFR < 45 ml/min/1.73 m^2				eGFR ≥ 45 and < 60 ml/min/1.73 m^2				eGFR ≥ 60 ml/min/1.73 m^2				p-value of interaction
	Abbreviated DAPT (n= 277)	Prolonged DAPT (n=294)	Hazard ratio (95% CI)	p-value	Abbreviated DAPT (n=434)	Prolonged DAPT (n=423)	Hazard ratio (95% CI)	p-value	Abbreviated DAPT (n=1584)	Prolonged DAPT (n=1567)	Hazard ratio (95% CI)	p-value	
NACE	37 (13.41)	39 (13.28)	1.01 (0.64-1.58)	0.970	38 (8.80)	44 (10.42)	0.84 (0.54-1.29)	0.416	97 (6.15)	99 (6.35)	0.96 (0.73-1.27)	0.792	0.814
MACCE	32 (11.59)	33 (11.24)	1.04 (0.64-1.69)	0.875	31 (7.18)	37 (8.76)	0.81 (0.50-1.30)	0.385	75 (4.76)	68 (4.37)	1.09 (0.78-1.51)	0.610	0.593
MCB	18 (6.69)	35 (12.10)	0.53 (0.30-0.93)	0.026	33 (7.73)	33 (7.91)	0.97 (0.60-1.57)	0.893	97 (6.19)	143 (9.23)	0.66 (0.51-0.85)	0.001	0.232
Death	19 (6.90)	25 (8.52)	0.80 (0.44-1.46)	0.474	20 (4.64)	21 (4.97)	0.93 (0.50-1.71)	0.806	36 (2.28)	35 (2.25)	1.01 (0.64-1.62)	0.952	0.835
Cardiovascular Death	12 (4.41)	16 (5.53)	0.79 (0.38-1.68)	0.547	7 (1.65)	8 (1.91)	0.85 (0.31-2.35)	0.756	18 (1.15)	20 (1.29)	0.89 (0.47-1.68)	0.714	0.975
Non-cardiovascular Death	4 (1.50)	5 (1.76)	0.85 (0.23-3.16)	0.805	10 (2.35)	10 (2.42)	0.97 (0.40-2.34)	0.950	15 (0.96)	13 (0.84)	1.14 (0.54-2.39)	0.733	0.919
Undetermined Death	3 (1.11)	4 (1.42)	0.79 (0.18-3.53)	0.756	3 (0.71)	3 (0.72)	0.97 (0.20-4.82)	0.972	3 (0.19)	2 (0.13)	1.48 (0.25-8.86)	0.668	0.869
Cerebrovascular event	5 (1.86)	7 (2.47)	0.76 (0.24-2.38)	0.632	3 (0.70)	8 (1.94)	0.36 (0.10-1.37)	0.134	9 (0.58)	17 (1.10)	0.52 (0.23-1.17)	0.114	0.710
Stroke	4 (1.48)	5 (1.77)	0.85 (0.23-3.17)	0.812	1 (0.23)	6 (1.45)	0.16 (0.02-1.34)	0.091	7 (0.45)	12 (0.78)	0.58 (0.23-1.46)	0.245	0.422
TIA	1 (0.38)	2 (0.71)	0.52 (0.05-5.77)	0.597	2 (0.47)	2 (0.48)	0.97 (0.14-6.91)	0.979	2 (0.13)	5 (0.32)	0.39 (0.08-2.03)	0.266	0.784
Myocardial infarction	13 (4.85)	8 (2.81)	1.74 (0.72-4.19)	0.220	14 (3.29)	14 (3.37)	0.97 (0.46-2.03)	0.936	33 (2.12)	27 (1.75)	1.21 (0.73-2.01)	0.466	0.610
Definite or probable ST	4 (1.50)	2 (0.70)	2.11 (0.39-11.53)	0.388	2 (0.48)	1 (0.24)	1.95 (0.18-21.50)	0.586	8 (0.51)	6 (0.39)	1.32 (0.46-3.80)	0.610	0.879
BARC bleeding													
BARC 1	11 (4.09)	17 (5.91)	0.67 (0.32-1.44)	0.307	6 (1.40)	17 (4.08)	0.34 (0.13-0.86)	0.022	48 (3.06)	75 (4.84)	0.63 (0.44-0.90)	0.011	0.449
BARC 2	9 (3.36)	21 (7.28)	0.44 (0.20-0.97)	0.041	21 (4.92)	23 (5.55)	0.88 (0.49-1.60)	0.684	72 (4.60)	108 (6.98)	0.65 (0.48-0.87)	0.004	0.375
BARC 3	10 (3.73)	11 (3.85)	0.96 (0.41-2.26)	0.923	13 (3.06)	10 (2.40)	1.26 (0.55-2.88)	0.578	30 (1.92)	38 (2.46)	0.78 (0.48-1.25)	0.298	0.594

BARC 3a	6 (2.23)	6 (2.11)	1.06 (0.34-3.28)	0.924	5 (1.18)	9 (2.16)	0.54 (0.18-1.60)	0.266	15 (0.96)	15 (0.97)	0.99 (0.48-2.02)	0.968	0.613
BARC 3b	4 (1.51)	4 (1.38)	1.06 (0.27-4.24)	0.933	6 (1.42)	0 (0.00)	12.67 (0.72-224.21)	0.031	11 (0.70)	16 (1.04)	0.68 (0.31-1.46)	0.320	0.581
BARC 3c	0 (0.00)	1 (0.37)	0.35 (0.01-8.56)	-	3 (0.71)	1 (0.24)	2.91 (0.30-28.00)	0.355	4 (0.26)	7 (0.45)	0.56 (0.16-1.92)	0.360	-
BARC 4	0 (0.00)	0 (0.00)	-	-	0 (0.00)	0 (0.00)	-	-	0 (0.00)	0 (0.00)	-	-	-
BARC 5	1 (0.39)	3 (1.06)	0.35 (0.04-3.38)	0.365	1 (0.24)	1 (0.24)	0.98 (0.06-15.59)	0.986	0 (0.00)	4 (0.26)	0.11 (0.01-2.04)	0.061	0.578
BARC 5a	0 (0.00)	1 (0.34)	0.35 (0.01-8.56)	1.000	0 (0.00)	0 (0.00)	-	-	0 (0.00)	1 (0.07)	0.33 (0.01-8.09)	0.497	-
BARC 5b	1 (0.39)	2 (0.72)	0.53 (0.05-5.80)	0.599	1 (0.24)	1 (0.24)	0.98 (0.06-15.59)	0.986	0 (0.00)	3 (0.19)	0.14 (0.01-2.71)	0.123	0.745
BARC 3 or 5	11 (4.11)	14 (4.88)	0.83 (0.38-1.82)	0.639	14 (3.30)	11 (2.63)	1.24 (0.56-2.73)	0.597	30 (1.92)	42 (2.71)	0.70 (0.44-1.12)	0.138	0.479

Abbreviations: BARC, Bleeding Academic Research Consortium; CI, confidence interval; CKD, chronic kidney disease; MACCE, major adverse cardiac and cerebral events; MCB, major or clinically relevant non-major bleeding; NACE, net adverse clinical events; ST, stent thrombosis; TIA, transient ischemic attack.

Nr of first events of each type (Kaplan-Meier failure %). Hazard ratio (95% CI) from Cox's time-to-first event analyses in ITT population. Continuity corrected risk ratios (95% CI) in case of zero events with Fisher's exact test p-value. Interaction p-value testing for modifying effect of CKD (yes or no) on the hazard ratio scale.



Supplementary Figure 1. Kaplan-Meier curves for all-cause mortality (A), myocardial infarction (B), stroke (C), and BARC Type 3 or 5 bleeding (D).

Abbreviations: BARC, Bleeding Academic Research Consortium; CI, confidence interval; DAPT, dual antiplatelet therapy; HR, hazard ratio.