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Coronary Chronic Total Occlusions Represent an Independent Predictor of Mortality in Ventricular Tachyarrhythmias

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Abstract

Aims: This study sought to assess the prognostic impact of coronary chronic total occlusions (CTO) in patients presenting with ventricular tachyarrhythmias on admission.

Methods and results: A large retrospective registry was used including all consecutive patients presenting with ventricular tachyarrhythmias on admission and undergoing coronary angiography from 2002 to 2016. Patients with a CTO were compared with all other patients (non-CTO) for prognostic outcomes. Statistics comprised Kaplan Meier and Cox regression analyses.

Within a total of 1,461 included consecutive patients with ventricular tachyarrhythmias on admission, a CTO was present in 20%. At mid-term follow-up of 18 months, the primary endpoint all-cause mortality had occurred in 46% of CTO patients compared to 27% of non-CTO patients (HR = 1.563; 95% CI 1.263 – 1.934; p=0.001). The rates of secondary endpoints were higher for in-hospital all-cause mortality at index (29% versus 20%, log rank p=0.027) and the composite endpoint of cardiac death at 24 hours, recurrent ventricular tachyarrhythmias and appropriate ICD therapies at mid-term follow-up (28% versus 20%; log rank p=0.005). Mortality rates were highest in CTO patients with stable CAD, acute myocardial infarction and in patients surviving index hospitalization.

Conclusion: In patients presenting with ventricular tachyarrhythmias on admission, the presence of a coronary CTO is independently associated with an increase of mid-term all-cause mortality, in hospital all-cause mortality and the composite endpoint of early cardiac death, recurrent ventricular tachyarrhythmias and appropriate ICD therapies.

Condensed abstract

This study retrospectively examined the prognostic impact of coronary chronic total occlusions (CTO) on survival in 1,461 patients admitted with ventricular tachyarrhythmias. The presence of a coronary CTO was independently associated with higher mid-term all-cause (HR=1.563; 95% CI 1.263–1.934; p=0.001), index in-hospital all-cause mortality (29% versus 20%, log rank p=0.027) and the composite endpoint of cardiac death at 24 hours, recurrent ventricular tachyarrhythmias and appropriate ICD therapies at mid-term follow-up (28% versus 20%; log rank p=0.005).

Abbreviations

ACE, angiotensin converting enzyme.

AMI, acute myocardial infarction.

ARB, angiotensin receptor blocker.

AVRD, arrhythmogenic right ventricular dysplasia.

CAD, coronary artery disease.

CCM, cardiac contractility modulation.

CPU, chest pain unit.

CRT-D, cardiac resynchronization therapy with defibrillator.

cMRI, cardiac magnetic resonance imaging.

CABG, coronary artery bypass grafting.

CPR, cardiopulmonary resuscitation.

CTO, chronic total occlusion.

CX, left circumflex.

ECG, electrocardiogram.

HR, hazard ratio.

ICD, implanted cardioverter defibrillator.

ICU, intensive care unit.

IQR, interquartile range.

J-CTO, Japanese CTO-score.

LVEF, left ventricular ejection fraction.

MVD, multi-vessel disease.

PCI, percutaneous coronary intervention.

RCA, right coronary artery.

SCA, sudden cardiac arrest.

SCD, sudden cardiac death-

SEM, standard error of mean.

NSTEMI/STEMI, (non-) ST segment elevation myocardial infarction.

s-ICD, subcutaneous ICD.

UMM, University Medical Centre Mannheim.

VF, ventricular fibrillation.

VT, ventricular tachycardia.

Introduction

In up to 80% of patients ventricular tachyarrhythmias and sudden cardiac death (SCD) are caused by acute myocardial infarction (AMI) and associated with known or undiagnosed coronary artery disease (CAD) and its complications. About half of cardiac deaths after AMI are related to ventricular tachyarrhythmias, such as ventricular tachycardia (VT) or fibrillation (VF).[1] The concept that ventricular tachyarrhythmias or SCD are potentially the corollary of risk of coronary chronic total occlusion (CTO) has only been looked at in few clinical studies.[2-5] However, these studies evaluated the role of CTO on ventricular tachyarrhythmias and SCD in preselected and different populations, such as ischemic cardiomyopathy, out-of-hospital cardiac arrest survivors and patients with an implanted cardioverter defibrillator (ICD).[4, 6, 7] CTOs are seen in about 18.4% of patients undergoing coronary angiography and may be linked to previous AMI or progressive CAD.[8] CTO may either induce acute myocardial ischemia, varying post-AMI myocardial scaring, as well as peri-infarct changes including hibernating myocardium supplied by developing collateral connections, or adverse remodeling with myocardial dilation and dysfunction.[9] These patho-anatomic changes within the border zones may sustain an abnormal myocardial electrical milieu, which is characterized by slow inhomogeneous activation, ventricular repolarization heterogeneity and ectopic activity facilitating the development of ventricular tachyarrhythmias and consecutive cardiac death. Evaluation of the association of CTOs as a potential trigger for ventricular tachyarrhythmias and early cardiac death is of great clinical interest. Therefore, this study evaluates the prognostic impact of CTO in patients presenting with ventricular tachyarrhythmias on admission.

Methods

Study patients, design and data collection

The present study included retrospectively all patients presenting consecutively with ventricular tachyarrhythmias on hospital admission and undergoing coronary angiography from 2002 until 2016 at one institution. Using the electronic hospital information system, all relevant clinical data related to the index event were documented. The present study is derived from an analysis of the "Registry of Malignant Arrhythmias and Sudden Cardiac Death - Influence of Diagnostics and Interventions (RACE-IT)" and represents a single-center retrospective registry including consecutive patients presenting with ventricular tachyarrhythmias and aborted cardiac arrest being acutely admitted to the University Medical Center Mannheim (UMM), Germany (clinicaltrials.gov identifier: NCT02982473) from 2002 until 2016. The registry was carried out according to the principles of the declaration of Helsinki and was approved by the medical ethics committee II of the Medical Faculty Mannheim, University of Heidelberg, Germany. Also see supplemental online information.

Definition of study groups, inclusion and exclusion criteria

For the present analysis, only patients presenting with ventricular tachyarrhythmias and with coronary angiography at index hospital stay were included in order to ensure reliable documentation of the presence of CAD and CTOs. A coronary CTO was defined according to the European consensus document. [10, 11] All coronary angiograms and reports were reassessed post-hoc by two independent interventional cardiologists specialized in CTO-PCI for the present data analysis.

The CTO group comprised all patients with a native un-revascularized CTO in coronary vessels with a diameter >1.5 mm. Identification of CTO and CAD was based on the judgment of the investigating interventional cardiologist during routine care. The CTO group also

included patients with acute revascularization of non-CTO-vessel by PCI or CABG. Moreover, patients with an occluded bypass graft on the CTO vessel were allocated to CTO group.

The control non-CTO group included all patients without CAD or CAD without a CTO, including CABG without a concomitant CTO and patients with acute PCI or CABG of non-CTO vessels. Patients with successfully revascularized CTOs either by CABG or PCI prior to ventricular tachyarrhythmias or SCD were included to the non-CTO group. Also, patients with successful CTO-revascularization during index hospitalization either by PCI or CABG were included in the non-CTO-group.

Overall exclusion criteria comprised patients without coronary angiography and coronary angiography after index hospital stay, patients without complete follow-up data regarding mortality and patients without documented ventricular tachyarrhythmias at index.

Study endpoints

The primary prognostic outcome was all-cause mortality at mid-term follow-up. Secondary endpoints were cardiac death occurring <24 hours after onset of ventricular tachyarrhythmias or an assumed unstable cardiac condition on admission, in-hospital all-cause mortality at index and the composite endpoint of cardiac death at 24 hours, first recurrent sustained ventricular tachyarrhythmias and first appropriate ICD therapies.

Overall follow-up period lasted until 2016. All-cause mortality was documented using our electronic hospital information system and by directly contacting state resident registration offices (bureau of mortality statistics) across Germany.

Statistical methods

Quantitative data are presented as mean ± standard deviation, median and interquartile range (IQR), and ranges depending on the distribution of the data and were compared using the Student's *t* test for normally distributed data or the Mann-Whitney *U* test for nonparametric data. Deviations from a Gaussian distribution were tested by the Kolmogorov-Smirnov test. Spearman's rank correlation for nonparametric data was used to test univariate correlations. Qualitative data are presented as absolute and relative frequencies and compared using the Chi^2 test or the Fisher's exact test, as appropriate. Uni- and multivariable Cox regression models were applied and hazard ratios (HR) were given together with 95% confidence intervals. Cox regression models were applied in the entire cohort. Multivariable Cox regression models were developed using the "backward selection" option, where only statistically significant variables ($p<0.05$) were included and analyzed simultaneously. Follow-up periods for evaluation of all-cause mortality were set when occurring within the individual index hospitalization (=in-hospital) and at 18 months (mid-term) according to the median survival of CTO patients to guarantee longest and completed follow-up of at least 50% of patients. Patients not meeting mid-term follow-up were censored. The result of a statistical test was considered significant for $p < 0.05$ and SAS, release 9.4 (SAS Institute Inc., Cary, NC, USA) was used for statistics.

Results

Study population

A total of 1,461 consecutive patients with ventricular tachyarrhythmias on admission at our institution underwent coronary angiography. At least one CTO of a major coronary artery was found in 20% of patients. The CTO was most commonly located in the right coronary artery (RCA) in 42%, followed by the left anterior descending artery (LAD) in 17% and circumflex (CX) in 19%, whereas 22% of patients had multiple CTOs **(Figure 1B)**. CTO patients were more likely to suffer from diabetes, hyperlipidaemia, NSTEMI, chronic kidney disease and were more frequently treated with amiodarone and digitalis **(Table 1)**. CTO patients revealed a higher rate of LVEF<35% alongside a higher rate of ICDs compared to non-CTO patients (47% versus 40%).

Distribution of CAD in patients with ventricular tachyarrhythmias

As shown in **supplemental table 1**, 33% of non-CTO patients revealed no evidence of CAD, whereas CTO patients were more likely to have multi-vessel coronary artery disease and prior CABG. Non-CTO-PCI was more common in non-CTO compared to CTO patients (46% versus 39%, p=0.033) and PCI in bypass grafts was rarely performed. CTO revascularization was performed rarely in 14 patients (allocated to the non-CTO group) (9 with CTO-PCI and 5 with CTO-CABG). The median J-CTO score was 2.8 (IQR 1.8-3.6) in CTO patients presenting with ventricular tachyarrhythmias.

Prognostic impact of CTO

At mid-term follow-up (18 months, IQR 5 days to 5.7 years), CTO patients had a higher all-cause mortality compared to non-CTO patients (mortality rate 40% versus 27%; log rank p=0.001; HR = 1.563; 95% CI 1.263 – 1.934; p=0.001) **(Figure 2, left panel)**, which was still

evident after multivariable adjustment (HR=1.412; 95% CI 1.081-1.843; p=0.011) (**Figure 3; left panel**).

The presence of a CTO was still univariably associated with increased mid-term all-cause mortality in the subgroups of females (HR=2.072; p=0.001), males (HR=1.437; p=0.004), VT (HR=1.505; p=0.028) VF (HR=1.614; p=0.001), LVEF ≥35% (HR=1.792; p=0.002), non-ICD (HR=1.977; p=0.001), AMI (HR=1,424; p=0.017) and stable CAD patients (HR=1.540; p=0.013) (data not shown).

Regarding secondary endpoints, CTO patients revealed higher rates of in-hospital all-cause mortality at index (29% vs. 20%, HR=1.032-1.701; p=0.027) **(Table 2)** and higher rates of the composite endpoint of cardiac death at 24 hours, recurrent ventricular tachyarrhythmias and appropriate ICD therapies at mid-term follow-up compared to non-CTO patients (28% versus 19%, log rank p=0.005, HR=1.418, 95% CI 1.102-1.825; p=0.007) **(Figure 2 right panel, Table 2)**. Associations of a CTO with the secondary composite endpoint were still univariably seen in the subgroups of males (HR=1.398; p=0.025), VT (HR=1.491; p=0.028), LVEF ≥35% (HR=1.721; p=0.012), non-ICD (HR=1.566; p=0.013) and stable CAD (HR=1.507; p=0.029) (data not shown).

Discussion

The present study evaluates the prognostic impact of coronary CTOs in consecutive patients presenting with ventricular tachyarrhythmias on admission. This real-world data suggests that coronary CTOs are common findings in up to 20% of patients presenting with ventricular tachyarrhythmias. The presence of at least one CTO is an important prognostic factor, associated with an increased risk of mid-term all-cause mortality, in-hospital all-cause mortality at index and the composite endpoint of cardiac death at 24 hours, recurrent ventricular tachyarrhythmias and appropriate ICD therapies. The adverse prognostic value of a CTO was still evident in the subgroups of patients with stable CAD and AMI, as well as after multivariable adjustment even in the entire study cohort. Therefore, this study consistently identifies the presence of a native un-revascularized CTO as a robust predictor of adverse outcomes in patients presenting with ventricular tachyarrhythmias on admission.[1]

Recent data showed that sustained monomorphic VT in patients with previous AMI were less likely to be affected by coronary revascularization.[1] Furthermore, myocardial revascularization appeared to be unlikely to prevent recurrent SCD in patients with extensive myocardial scarring as well as in the presence of a markedly depressed LVEF.[1] Hence, VT was shown to be still inducible after surgical revascularization in survivors of cardiac arrest in 80% of patients with multi-vessel CAD with a prevalence of VT and VF at index of 66%. Accordingly, VT recurrence occurred in post-AMI patients after successful revascularization either by PCI or CABG even in the presence of LV dysfunction below 30%. Notably, VT or VF were still inducible in CAD patients independently of a complete or incomplete revascularization by PCI or CABG. In contrast, catheter ablation only proved efficacy to reduce the rates of VT recurrence (with a higher recurrence rate in patients with CTO as compared to non-CTO patients)[2], ICD therapy and symptom-free follow-up, but has never proven any better survival in patients presenting with VT and CAD.[1]

Whether CTO might be associated with development of ventricular tachyarrhythmias has so far been investigated in a small number of retrospective observational studies. The VACTO studies demonstrated significantly higher rates of mortality and ventricular tachyarrhythmias in 71 native CTO patients compared 91 patients without CTO, and revealed a CTO being an independent predictor for appropriate ICD therapy both in ICD carriers for primary and secondary prevention.[3, 5] In contrast, Raja et al. found no difference in mortality or ICD therapy for sustained VT between ICD patients with native CTO, revascularized CTO (total CTO patients n=213) and without CTO (n=95). Notably in this study the absence of a CTO in the left circumflex was protective with regard to ventricular tachyarrhythmias.[4] Surprisingly, the presence of a CTO in the territory of a previous AMI was shown to be associated with a higher rate of VT recurrence after VT ablation, as well as repeat ablation rate.[2] In contrast, the major strength of the present study consists in the consecutive recruitment of patients with ventricular tachyarrhythmias and CTO straight from the admission scenario, where the adverse prognostic impact of a CTO was both demonstrated in the initially admitted study cohort and in those patients surviving and discharged from index hospitalization.

Di Marco et al. identified the presence of larger border zones in patients with an infarct-related CTO artery, alongside with reduced LVEF and renal impairment, as significant predictors for VT recurrence in 47 patients with and 37 patients without infarct-related CTO arteries.[2, 7] VF originates mostly from an ischemic substrate, whereas VT represents a scar-related substrate in the presence of ischemic cardiomyopathy with a prior history of AMI, or in the context of structural or inflammatory heart disease.[1] Additionally, mechanisms such as early reperfusion, reduction of scars and infarct size, limitation of infarct expansion, recovery of function and improved collateral flow may lead in turn to better electrical stability within the myocardium.[9] It may be speculated that the presence of a CTO may adversely accentuate

above effects and may - unless revascularized - sustain a milieu of permanent electrical instability.

Study limitations

This observational and retrospective registry-based analysis reflects a realistic picture of consecutive health-care supply of high-risk patients presenting with ventricular tachyarrhythmias and SCD right on hospital admission. The total number of patients with LVEF <35% and surviving index hospitalization without further implantation of an ICD was low (19%, n=73), which may be attributed to improvement of LVEF above 35% at follow-up in major part and lack of data documentation in minor part. Myocardial scarring was not assessed within the present study for instance by cardiac MRI in all patients. Therefore, the prognostic benefit of potential "scar-related" responders to CTO revascularization compared to non-responders was not assessed. Furthermore, rates of CTO revascularization were very low (only 5%) and reliable conclusions about its prognostic effect in patients presenting with ventricular tachyarrhythmias are not possible.

Conclusions

In patients presenting with ventricular tachyarrhythmias on admission, the presence of a coronary CTO is independently associated with an increase of mid-term all-cause mortality, in hospital all-cause mortality and the composite endpoint of early cardiac death, recurrent ventricular tachyarrhythmias and appropriate ICD therapies.

Impact on daily practice

In high-risk patients presenting with ventricular tachyarrhythmias on admission undergoing coronary angiography the presence of a native un-revascularized CTO represents

a consistently robust predictor of mid-term all-cause mortality, irrespective of stable CAD or acute myocardial infarction, as well as of in-hospital index all-cause mortality and the composite endpoint of early cardiac death, recurrent ventricular tachyarrhythmias and appropriate ICD therapies.

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Conflict of interest

The authors declare that they do not have any conflict of interest.

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Figures Legends

Figure 1A: Flow-chart of patient selection.

Figure 1B: Distribution of location of CTOs.

Figure 2: Presence of a coronary CTO was associated with increased rates of the primary endpoint all-cause mortality at mid-term follow-up **(left panel)** and the composite endpoint of cardiac death at 24 hours, recurrent ventricular tachyarrhythmias and appropriate ICD therapies at 18 months **(right panel)**.

Figure 3: In multivariable Cox regressions, the presence of a coronary CTO was still associated with the primary endpoint all-cause mortality at mid-term follow-up (after 2 steps of backward elimination) **(left panel)**, as well as with the composite endpoint of cardiac death at 24 hours, recurrent ventricular tachyarrhythmias and appropriate ICD therapies at mid-term follow-up (after 6 steps of backward elimination) **(right panel)**.

Table 1. Study population.

Characteristic	non-CTO (n=1,171; 80%)	CTO (n=290; 20%)	p value
Gender, n (%)			
Male	863 (74)	229 (79)	0.065
Age, median (range)	65 (15-91)	70 (39-89)	**0.001**
Ventricular tachyarrhythmia, n (%)			
Ventricular tachycardia	622 (53)	154 (53)	0.997
Non-sustained	369 (61)	76 (50)	**0.011**
Sustained	232 (39)	76 (50)	

Monomorphic	587	(98)	148	(97)	0.828
Polymorphic	14	(2)	4	(3)	
Slow	6	(1)	3	(2)	0.323
Fast	595	(99)	149	(98)	
Induced	220	(37)	55	(36)	0.923
Electrical storming	10	(2)	3	(2)	0.768
Torsade de pointes	8	(1)	0	(0)	0.368
Not documented	21	-	2	-	-
VT cycle length, median (IQR)	305 (280-340)		325 (300-345)		0.486

Ventricular fibrillation	549	(47)	136	(47)	0.997
Cardiopulmonary resuscitation	569	(49)	142	(49)	0.909
In hospital	364	(31)	80	(28)	0.246
Out of hospital	205	(18)	62	(21)	0.127
Cardiovascular risk factors, n (%)					
Arterial hypertension	704	(60)	191	(66)	0.072
Diabetes mellitus	279	(24)	106	(37)	**0.001**
Hyperlipidemia	341	(29)	123	(42)	**0.001**
Smoking	379	(32)	93	(32)	0.923

Cardiac family history	137	(12)	30	(10)	0.516
Comorbidities at index, n (%)					
Acute myocardial infarction	461	(39)	122	(42)	0.400
STEMI	190	(16)	24	(8)	**0.001**
NSTEMI	271	(23)	98	(34)	**0.001**
Cardiogenic shock	209	(18)	60	(21)	0.264
Atrial fibrillation	324	(28)	81	(28)	0.929
Dilated cardiomyopathy	58	(5)	0	(0)	**0.001**
Hypertrophic cardiomyopathy	6	(0.5)	0	(0)	0.605

ARVD	7	(0.6)	0	(0)	0.358
Takotsubo CMP	7	(0.6)	0	(0)	0.358
Myocarditis	4	(0.3)	0	(0)	1.000
Brugada	11	(0.9)	0	(0)	0.098
Long QT	29	(3)	4	(1)	0.260
Intracranial hemorrhage	6	(0.5)	0	(0)	0.222
Anemia	70	(6)	16	(6)	0.765
Chronic kidney disease	524	(46)	170	(60)	**0.001**
Liver cirrhosis	8	(0.7)	2	(0.7)	0.990

Respiratory Failure	7	(0.6)	5	(2)	0.057
Intoxication (drugs, medication)	6	(0.5)	1	(0.3)	1.000
Neurological	35	(3)	7	(2)	0.600
Systemic Infection	24	(2)	12	(4)	**0.040**
Electrolyte disorder	7	(0.6)	3	(1)	0.419

Left ventricular ejection function, n (%)

LVEF ≥55%	336	(29)	36	(12)	
LVEF 54-35%	330	(28)	91	(31)	**0.001**
LVEF <35%	269	(23)	115	(40)	

Not documented	236	(20)	48	(17)	-
Electrical therapies, n (%)					
Electrophysiological examination	319	(27)	64	(22)	0.073
Ablation therapy	49	(4)	6	(2)	0.090
Patients at discharge, n (%)	937	(82)	206	(71)	**0.001**
Medication at discharge, n (%)					
Beta-blocker	786	(84)	179	(87)	0.281
ACE-inhibitor/ ARB	730	(78)	172	(84)	0.075
Aldosterone antagonist	80	(9)	25	(12)	0.105

Statin	623	(67)	172	(84)	**0.001**
Amiodarone	108	(12)	50	(24)	**0.001**
Digitalis	92	(10)	34	(17)	**0.006**
Overall ICDs, n (%)	466	(50)	137	(67)	**0.001**
ICD	416	(89)	128	(93)	**0.007**
CRT-D	37	(8)	7	(5)	0.506
s-ICD	13	(3)	2	(2)	0.525
Follow up times					
Hospitalization total; days (median (IQR))	11 (6-19)		13 (6-24)		**0.001**

ICU time; days (median (IQR))	5 (3-10)	4 (1-10)	**0.001**
Follow-up; days (mean; median (range))	1520;1176 (0-5106)	1521; 1176 (0-5106)	**0.001**

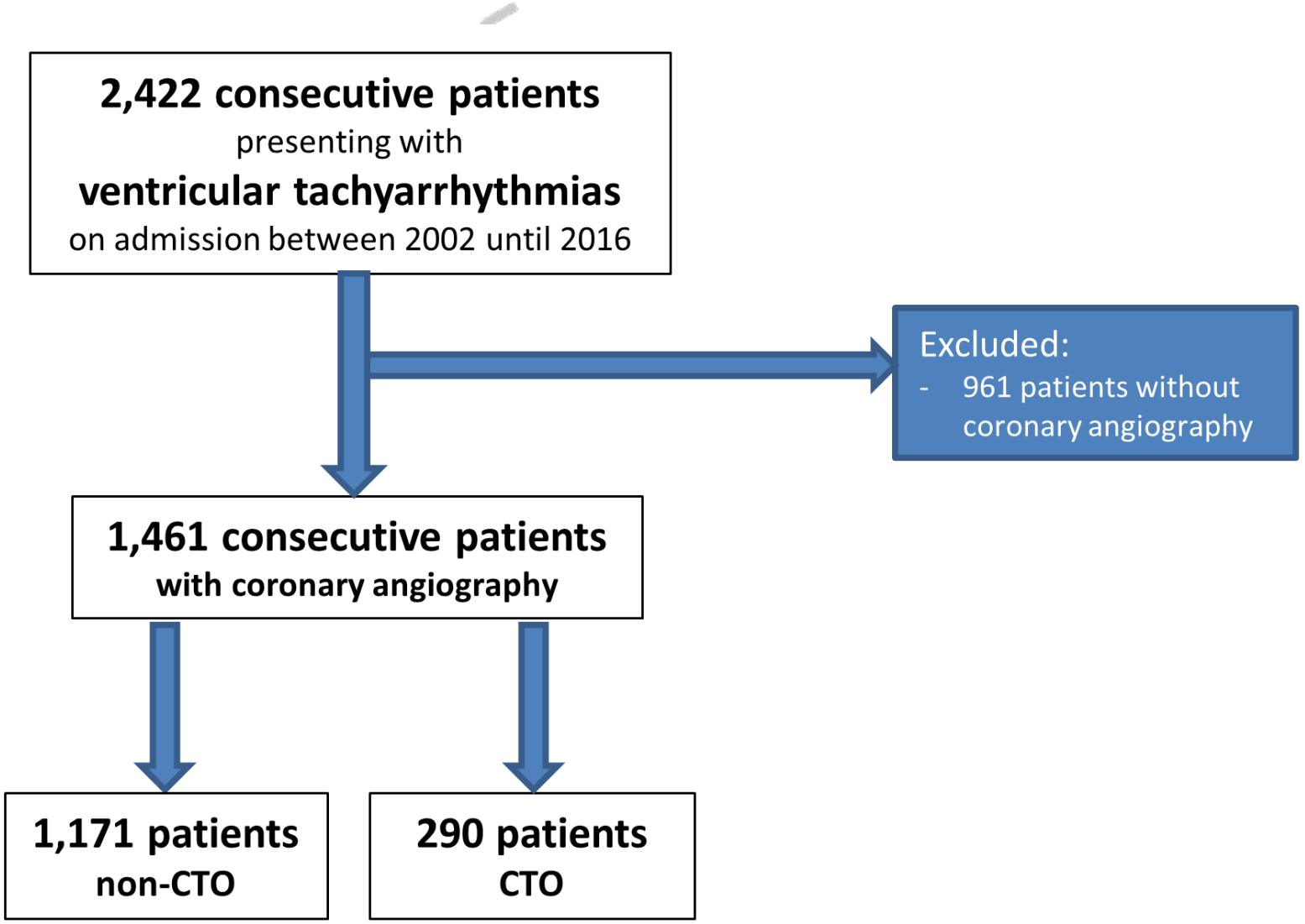
ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; ARVD, arrhythmogenic right ventricular dysplasia; CMP, cardiomyopathy; CRT-D, cardiac resynchronization therapy plus defibrillator; ICD, implantable cardioverter-defibrillator; ICU, invasive care unit; IQR, interquartile range; LVEF, left ventricular ejection fraction; (N)STEMI, (non-)ST segment myocardial infarction, VT, ventricular tachycardia.

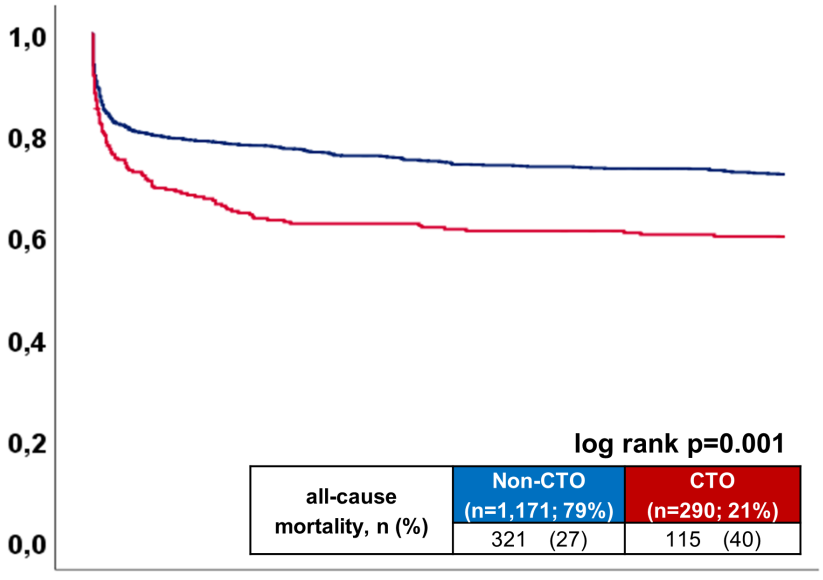
Table 2. Primary and secondary endpoints.

Characteristic	non-CTO (n=1,171; 79%)		CTO (n=290; 21%)		HR	95% CI	P value
Primary endpoint, n (%)							
All cause-mortality, at 18 months	321	(27)	115	(40)	1.56	1.263-[illegible]	**0.001**
Secondary endpoints, n (%)							
Cardiac death, at 24 hours	110	(9)	38	(13)	1.45	0.981-[illegible]	0.062
All-cause mortality, at index hospitalization	234	(20)	84	(29)	1.32	1.032-[illegible]	**0.027**

Combined endpoint (cardiac death at 24 hours, recurrent ventricular tachyarrhythmias, appropriate ICD therapies at 18 months)	236	(20)	81	(28)	1.41	1.102-	**0.007**

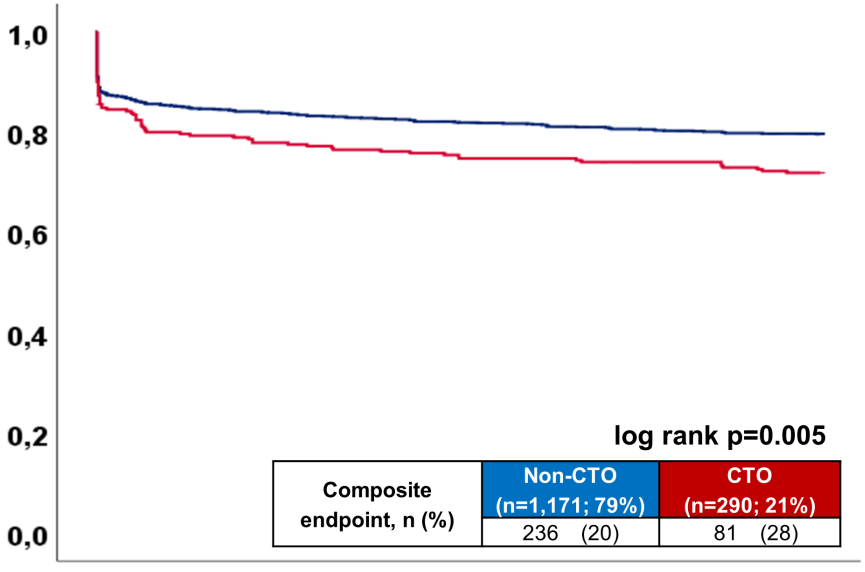
CI, confidence interval; HR, hazard ratio; ICD, implantable cardioverter defibrillator.





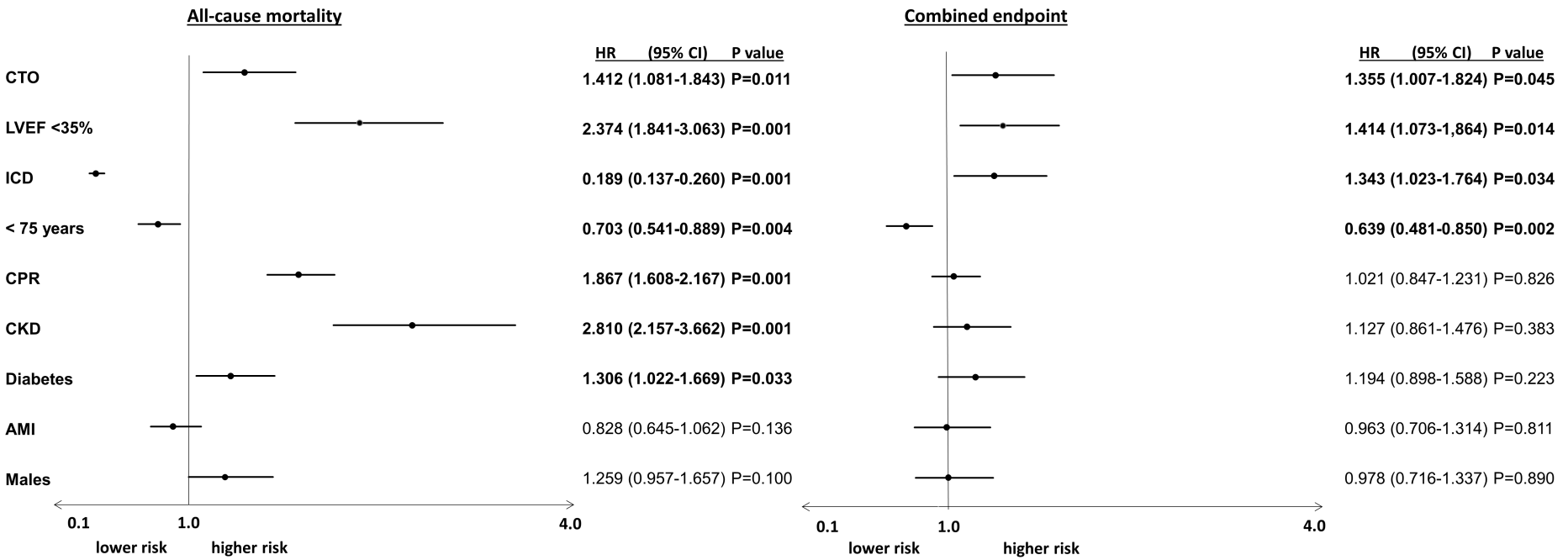
Patients at risk

Non-CTO	1171	909	871	838	817	797	769
CTO	290	192	176	174	172	167	165



Patients at risk

Non-CTO	1171	972	940	914	890	862	834
CTO	290	228	218	215	210	206	198



Supplemental Table 1. Distribution of coronary artery disease.

Characteristic	non-CTO (n=1,171; 80%)		CTO (n=290; 20%)		p value
Coronary artery disease, n (%)	789	(67)	290	(100)	**0.001**
No evidence of CAD	382	(33)	0	(0)	
1-vessel	285	(24)	49	(17)	
					0.001
2-vessel	254	(22)	84	(29)	
3-vessel	250	(21)	147	(54)	
Presence of CABG	136	(12)	57	(20)	**0.001**

Intracoronary thrombus	105	(9)	15	(5)	**0.035**
CTO lesions, n (%)					
RCA	-	-	122	(42)	-
LAD	-	-	49	(17)	-
LCx	-	-	55	(19)	-
Multiple CTOs	-	-	64	(22)	-
CTO lesion characteristics, n (%)					

Blunt stump	-	-	160	(55)	-
Bending >45°	-	-	165	(57)	-
Severe calcifications	-	-	113	(39)	-
CTO length ≥20mm	-	-	249	(86)	-
CTO length <20mm	-	-	41	(14)	-
J-CTO score, median (IQR)	-	-	2.8 (1.8-3.6)		-
J-CTO Score ≥3, n (%)	-	-	139	(48)	-
Collateral connections, n (%)					

Ipsilateral	-	-	232	(80)	-
Contralateral	-	-	247	(85)	-
Werner class					
CC0	-	-	5	(2)	-
CC1	-	-	215	(75)	-
CC2	-	-	70	(24)	-
Levin class					
Septal	-	-	281	(97)	-

Bridge	-	-	119	(41)	-
Epicardial	-	-	249	(86)	-
Revascularization types, n (%)					
CTO-PCI successful	9	(0.8)	-	-	-
Sent to CTO-CABG	5	(0.4)	-	-	-
CTO-PCI unsuccessful	-	-	4	(1)	-
Non-CTO-PCI	542	(46)	114	(39)	**0.033**
Bypass-PCI	7	(0.6)	1	(1)	1.000

Sent to non-CTO CABG	25	(2)	14	(5)	**0.011**
CPR during coronary angiography	80	(7)	24	(8)	0.392

CABG, coronary artery bypass grafting; CPR, cardiopulmonary resuscitation; LAD, left anterior descending; LCx, left circumflex; PCI,percutaneous coronary intervention; RCA, right coronary artery.