EuroIntervention

Causes of death in early MI survivors with persistent infarct artery occlusion: results from the Occluded Artery Trial (OAT)

Irene M. Lang[1], MD; Sandra A. Forman[2], MA; Aldo P. Maggioni[3], MD; Witold Ruzyllo[4], MD; Jean Renkin[5], MD; Carlos Vozzi[6], MD; P. Gabriel Steg[7], MD; Jose-Maria Hernandez-Garcia[8], MD, PhD; Krzysztof Zmudka[9], MD; Manuel Jimenez-Navarro[8], MD; George Sopko[10], MD, MPH; Gervasio A. Lamas[11], MD; Judith S. Hochman[12], MD

1. Medical University of Vienna, Vienna, Austria; 2. Maryland Medical Research Institute, Baltimore, MD, USA; 3. Italian Association of Hospital Cardiologists (ANMCO) Research Center, Florence, Italy; 4. National Institute of Cardiology, Warsaw, Poland; 5. Cliniques Universitaires Saint-Luc, Brussels, Belgium; 6. Instituto De Intervenciones Cardiovasculares S.A., Rosario, Argentina; 7. INSERM U-698, Université Paris 7 and AP-HP, Paris, France; 8. Hospital Universitario Virgen de la Victoria de Málaga, Malaga, Spain; 9. Jan Pawel II Hospital, International Cardiology Clinic, Krakow, Poland; 10. National Heart, Lung, And Blood Institute, Bethesda, MD, USA; 11. Mount Sinai Medical Center, Miami Beach, FL, USA; 12. New York University School of Medicine, New York, NY, USA

Abstract

Aims: OAT randomised patients with an occluded infarct artery three to 28 days after myocardial infarction (MI). The study demonstrated that PCI did not reduce the occurrence of the primary composite endpoint of death, re-MI, and New York Heart Association class IV heart failure in comparison with patients assigned to optimal medical therapy alone (MED). In view of prior literature in similar cohorts showing fewer sudden cardiac deaths and less left ventricular (LV) remodelling, but excess re-MI with PCI, causes of death were analysed in more detail.

Methods and results: Stepwise Cox regression was used to examine baseline variables associated with causes of death. The immediate and primary cause of death did not differ between 1,101 PCI and 1,100 MED patients. One-year cardiovascular death rates were 3.8% for the PCI group, and 3.7% for the MED group, and 0.9% per year for the next four years in both groups. Five of six cases of cardiac rupture occurred in patients undergoing PCI.

Conclusions: In stable post-MI patients with occlusion of the infarct-related artery, PCI did not change the rate or cause of death. The observation that the majority of cardiac ruptures occurred in patients undergoing PCI deserves further investigation.

* Corresponding author: Department of Internal Medicine II, Division of Cardiology, Medical University of Vienna, Währinger Gürtel 18-20, 1090 Vienna, Austria
E-mail: irene.lang@meduniwien.ac.at

Introduction

The Occluded Artery Trial (OAT) tested the hypothesis that stable, high-risk patients with persistent total occlusion of the infarct-related artery (IRA) benefit from percutaneous coronary intervention (PCI) with opening of the occlusion within three to 28 days after the index infarction. This "open artery hypothesis" has previously been addressed by studies demonstrating increased electrical stability with fewer sudden cardiac deaths (SCD)[1], less adverse LV remodelling with PCI[2-5], and the provision of vascular territories that function as collaterals in the instance of future events[6,7].

The primary endpoint of OAT was the first occurrence of recurrent myocardial infarction (MI), hospitalisation/short stay unit treatment of New York Heart Association class IV congestive heart failure (HF), or death. After randomising 2,166 patients to PCI vs. optimal medical therapy (MED), the cumulative primary event rate was 17.2% in the PCI group, and 15.6% in the medical treatment group, indicating that PCI did not reduce the composite endpoint[8]. The rate of death was not different between treatment groups after a mean follow up of 2.9 years, yet trends were observed towards excess reinfarction but improved LV remodelling in the PCI group in a mechanistic ancillary study[8]. The causes of death analysis in OAT had not been prespecified, and its purpose was to examine whether there were more MI-related deaths in the PCI group.

Methods

Study population

Patients were eligible for enrolment in OAT if coronary angiography, performed on days three to 28 after myocardial infarction, showed total occlusion of the IRA with poor or absent antegrade flow, defined as a Thrombolysis in Myocardial Infarction (TIMI) flow grade of 0 or 1, and if they met a criterion for increased risk, defined as an ejection fraction of less than 50% (assessed by echocardiography, radionuclide ventriculography, or contrast ventriculography), proximal occlusion of a major coronary artery with a large risk region, or both. Qualifying angiograms were reviewed at a core angiography laboratory[9]. The qualifying period of three to 28 days was based on calendar days; day one was the day of the onset of symptoms. Exclusion criteria were NYHA class III or IV heart failure, shock, a serum creatinine concentration higher than 2.5 mg per decilitre (221 μmol per litre), significant left main or three-vessel coronary artery disease, low threshold or rest angina, or severe ischaemia on stress testing, which was required if the infarct zone was not akinetic or dyskinetic.

Patient recruitment

During two, one-month periods, 4,623 patients with acute myocardial infarction not eligible for the Occluded Artery Trial were recorded in screening logs. Among those patients with ST elevation myocardial infarction logged, 63% underwent primary PCI[8]. Based on a broad literature search[10-16], it is estimated that in contemporary PCI services, approximately 109,000 patients per year would fulfil OAT eligibility criteria in the USA.

Definitions

The immediate cause of death, and disease or injury initiating the train of events leading directly or indirectly to death (primary cause of death) were examined on an intention-to-treat basis.

All deaths were categorised by an independent morbidity and mortality classification committee (MMCC, ≥ two reviewers per event), which was blinded to treatment assignments with reporting of the primary and immediate causes using standardised definitions.

Patient follow-up

For this analysis, we have updated the database using all patients enrolled through June 30, 2006 (N=2201) and included all follow-ups through March 30, 2007. Compared with the database underlying the primary OAT report[8], we have added 35 patients enrolled in an extension phase of the viability ancillary study in 2006. The average follow-up is now 3.2 years, compared with the original OAT cohort follow-up of 2.9 years.

Statistical analysis

Baseline characteristics of non-surviving patients were summarised as frequencies and percentages for categorical variables and as means and standard deviations for continuous variables. Comparisons by cause of death (cardiovascular death versus non-cardiovascular death, and sudden death versus non-sudden death) were performed using chi square/Fisher's exact test for categorical variables and Student t-test for continuous variables. Mantel-Haenszel chi-square was used for test of trend over time. Univariable and multivariable Cox proportional hazards models were developed to evaluate the relations between baseline characteristics and the occurrence of cardiovascular death, and separately for the occurrence of sudden death. Kaplan-Meier life-table event rates are presented graphically.

To minimise Type I errors, it was pre-specified by the study protocol that a p-value of less than 0.01 would be considered as showing evidence of differences in secondary analysis.

SAS version 9.1.3 (SAS Institute, Cary, NC, USA) was used for statistical analyses.

Results

Causes of death

Of 2,201 study patients, 1,101 had been randomly assigned to routine PCI plus optimal medical therapy, and 1,100 to optimal medical therapy alone. Death occurred in 184 patients. Analysis was based on 174 cases with available cause of death information (Table 1).

Of these, 42 patients had reached a component of the primary endpoint prior to death, including 24 patients with class IV heart failure, 11 patients with MI, and seven patients with both heart failure and MI. Patients who had a recurrent MI prior to death were more likely to die of recurrent MI (immediate death in 27.3%, primary cause of death in 40.9%). Frequent cause of death in patients with classes III or IV heart failure was heart failure class IV

Table 1. Adjudicated causes of death in 2,201 randomised patients.

	Immediate Cause		Primary Cause	
	PCI n	MED n	PCI n	MED n
MI	2	4	15	12
Stroke	0	2	0	4
Intracerebral haemorrhage	0	2	0	1
Worsening HF	6	4	6	9
Arrhythmia	8	9	2	1
Cardiac rupture	4	2	na	na
Sudden unexplained	35	33	33	28
Other cardiovascular	6	3	6	1
Non-cardiovascular	28	26	27	29
Missing	3	7	3	7
Total	92	92	92	92

(immediate death in 25%, primary cause of death in 41.7%). Sudden unexplained deaths occurred mainly in patients who had not had a previous event (40% of patients, compared with 26% of patients who had had a previous event). There were no statistically significant differences in the distribution of causes of death by treatment group (Table 1). During follow-up, 49 patients received implantable automated defibrillators (ICDs, 28 PCI assigned, 21 MED assigned patients). Primary cause of death in eight patients with AICD was worsening heart failure in four patients (two MED and two PCI assigned), sudden death in two (one after failed PCI, and one assigned to MED), and non-cardiovascular in two patients who had been assigned to PCI.

PCI-related death occurred in four PCI group patients and two MED patients undergoing PCI, with two patients dying from cardiac rupture, two from arrhythmia, and two patients dying from stent thromboses at six and eight days from implantation.

Baseline characteristics of patients by survival status

While baseline characteristics of the patients in the two treatment groups were similar except for a higher prevalence of diabetes in the group assigned to MED, baseline characteristics of patients suffering death versus survivors differed significantly with regard to age (62.7 [30–88] versus 58.2 [24–89] years, p<0.0001), previous PCI (9.8 versus 4.3%, p<0. 001), diabetes (31.5% versus 19.6%, p=0.0001), history of angina (34.8% vs. 21.4%, p<0.0001), history of hypertension (60.3% vs. 47.6%, p<0.001), history of cerebrovascular disease (8.7% vs. 3.3%, p<0.001), history of chronic heart failure (CHF, 10.9% vs. 1.6%, p<0.0001), pre-existing NYHA functional class (Classes >I in 33.7% compared with 19.7%, p<0.0001) and Killip classes (Classes II-IV in 37.7% compared with 17.3%, p<0.0001). Glomerular filtration rate at baseline was <60 ml/min/1.73 m^2 in 28.7% of patients eventually suffering death, compared with 13.5% of patients surviving (p<0.0001). Time-to-randomisation from index MI (9.8 [6.6 days]) versus 11.1 (7.8 days), (medians ±SD, P=0.03) was shorter in patients who died.

Anterior and lateral ST elevations were more common in non-survivors (anterior, p=0.01, lateral, p<0.01). These electrocardiographic findings were in accord with the angiographic findings of more frequent LAD occlusion in non-survivors (45.6% as compared with 35.2% in survivors, p=0.003). Collateral grades and TIMI flow in the IRA were similar in non-survivors and survivors. Multivessel disease was more frequent in non-survivors (25.5% versus 16.6%, p=0.002). LVEF assessed by echo (29%), radionuclide ventriculography (3.4%) or by left ventricular angiography (67.5%) at baseline was lower in non-survivors (40.8%±11.3%) compared with survivors (46.9%±10.6, p<0.0001).

There were higher rates of use of diuretics (37% vs. 15%, p<0.0001) at hospital discharge, digitalis (11% vs. 2% p<0.0001) and insulin (13% vs. 6% p<0.0001) in patients later suffering death. By contrast, use of lipid-lowering medication was more prevalent in survivors (dead, 71.2%, versus survivors, 82.2%). Use rates of beta blockers, angiotensin converting enzyme inhibitors and aldosterone antagonists were similar in patients who survived and those who did not, as were other medication use rates (data not shown).

Baseline characteristics of patients suffering non-cardiovascular compared with cardiovascular death (Table 2)

When patients were stratified into those suffering cardiovascular versus non-cardiovascular death, baseline characteristics did not differ between groups, with the exception of ST elevation during the index MI, which was more frequent in patients suffering cardiovascular death. Heart rate (77 versus 72 beats per minute, and blood glucose (136 versus 111 mg/dl) were also higher among those who died of cardiac causes. There were no differences in cardiovascular, antiplatelet and anti-diabetic medication use rates among non-survivors grouped by cause of death with the exception of digitalis, which was used far less in patients suffering sudden cardiac death in comparison with patients suffering non-sudden cardiac death (4.4% versus 14.4%).

There were no differences in the use of thrombolytic agents during the first 24 hours after onset of index MI (22.2% versus 23.3%, p=0.87) in patients suffering non-cardiovascular death versus patients suffering cardiovascular death (Table 2). By contrast, glycoprotein IIb/IIIa antagonists were employed more frequently in patients later suffering a cardiovascular death (p=0.03). However, bleeding was in no instance reported as a cause of death.

At angiography, occlusion of the LAD was observed more often in patients suffering cardiovascular death (52.5% vs. 29.6% p=0.005, Table 2). Multivessel disease was more frequent in patients suffering cardiovascular death (30.0% vs. 14.8%, p=0.03, Table 2). A left ventricular angiogram demonstrated lower ejection fractions in patients suffering cardiovascular death (39.7 vs. 46.1, p=0.002, Table 2).

Baseline characteristics stratified by immediate causes of death

The variables shown in Table 2 were examined for their relationship to immediate cause of death. Patients dying from heart failure had

Table 2. Baseline clinical and angiographic characteristics by non-cardiovascular versus cardiovascular cause of death.

Characteristic	Non CV-Death		CV Death		P
	N=54		N=120		
Clinical					
Age	64.1 (30 - 85)		61.9 (32 - 88)		0.27
Std Dev	12.5		11.7		
Gender	N	%	N	%	
Male	39	72.2	91	75.8	0.61
Race					
White	43	79.6	96	80.0	0.96
History of	N/Total	%	N/Total	%	P
Angina	17	31.5	42	35.0	0.65
Myocardial Infarction	8	14.8	18	15.0	0.97
Cerebrovascular disease	3	5.6	10	8.3	0.76
Peripheral vessel disease	7	13.0	8	6.7	0.17
Renal insufficiency	4	7.4	3	2.5	0.21
Hypertension	35	64.8	69	57.5	0.36
AICD	1	1.9	1	0.8	0.53
CHF	4	7.4	14	11.7	0.39
PCI	7	13.0	10	8.3	0.34
CABG	2	3.7	1	0.8	0.23
Cardiac risk factors					
Diabetes	15	27.8	39	32.5	0.53
Hypercholesterolaemia	24	44.4	60	50.0	0.50
Family history of CAD	17	31.5	45	37.5	0.44
Current cigarette smoker	19	35.2	36	30.0	0.50
Highest NYHA Classification					
Prior to index MI or					
At randomisation					
Class >I	14	25.9	42	35.0	0.24
Highest Killip class II-IV during					
Index MI	17	31.5	47/119	39.5	0.31
New Q waves > 0.03 sec and/or					
Q-wave voltage > 1/3 QRS					
in > two related leads on ECG	33	61.1	77	64.2	0.70
ST segment elevation	33	61.1	92/118	78.0	0.02
ST segment elevation or					
New Q-wave or ST segment					
Elevations	39	72.7	101	84.2	0.07
ST-segment elevation or Q-wave					
Or R-wave loss	43	79.6	103	85.8	0.30
Thrombolytic therapy					
During first 24hours after onset	12	22.2	28	23.3	0.87
Interval between MI and randomisation,	Days		Days		
Mean, SD	9.6(6.0)		10.0(7.0)		0.72
Median (25-75%)	8.0(4-13)		8.0(4-14)		
Infarct related artery (IRA)					
LAD	16	29.6	63	52.5	0.005
TIMI Flow grade in IRA					
Grade 0	45	83.3	101	84.2	
Grade I	9	16.7	18	15.0	
Grade II	0	0.0	1	0.8	
Grade III	0	0	0	0.0	0.77
Collateral vessels present					
Grades I-III	50	92.6	96/119	80.7	0.05
Multivessel disease	8	14.8	36	30	0.03
Vital parameters	**mean**	**SD**	**mean**	**SD**	**P**
Systolic blood pressure (mmHg)	120.2	18.8	118.9	20.4	0.67
Diastolic blood pressure (mmHg)	70.4	11.3	71.9	11.1	0.42
Pulse pressure (mmHg)	49.8	14.8	47.0	16.4	0.27
Estimated glomerular filtration rate (ml/min/1.73 m²)	67.0	18.0	75.4	23.1	0.02
Blood glucose level (mg/dL)	111.4	22.9	135.6	55.6	0.004
Heart rate (beats/min)	72.3	11.0	76.7	12.3	0.03
Ejection fraction	46.1	12.4	39.7	12.7	0.002
Body mass index (kg/m²)	29.9	7.2	28.9	6.3	0.36

highest Killip classes during the index MI, and the majority of these patients were in NYHA classes >I at randomisation. Patients suffering "other immediate cardiovascular death" were diabetic in roughly two thirds of cases. No statistical tests were performed because of the small size of subsets.

Timelines of causes of death

Sudden death and other cardiovascular death occurred mainly within the first six months after randomisation (p<0.001, Figure 1). Arrhythmia accounted for 10% of deaths each year over five years observation. MI and stroke occurred mainly over the initial two years of observation. In year five the leading cause of death was non-cardiovascular (7/14). Figure 2 demonstrates Kaplan-Meier event curves by treatment group for cardiovascular death and sudden death. No differences were observed according to assigned treatment.

Predictors of all-cause mortality and cardiovascular death

In multivariable analysis, independent predictors of death were Killip class>1 during index MI {HR 1.75, p<0.001}, history of
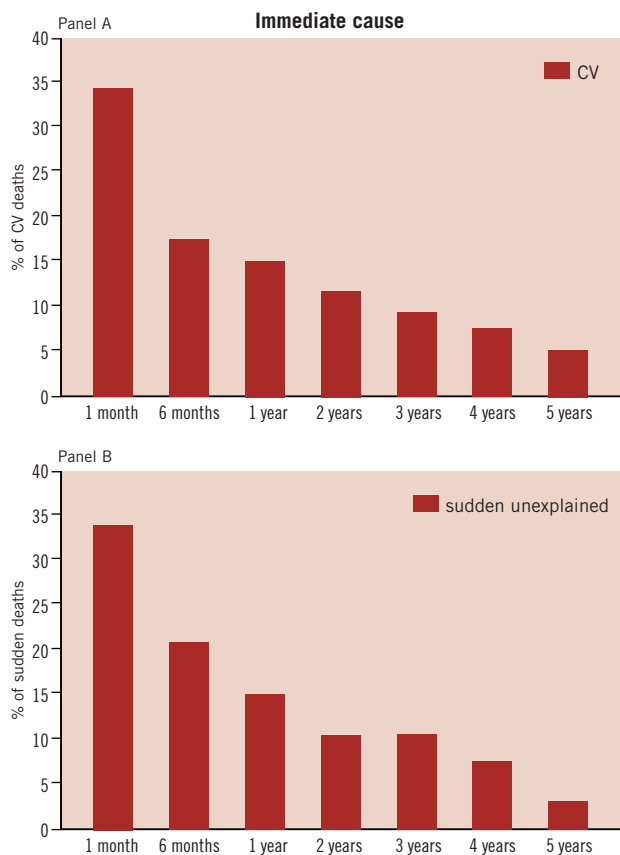

Figure 1. Timeline of occurrence of immediate cardiovascular (Panel A) and sudden death (Panel B). Panel A demonstrates the percent distribution of CV death for the 120 CV deaths. Panel B shows percent distribution of sudden death for the 68 sudden deaths
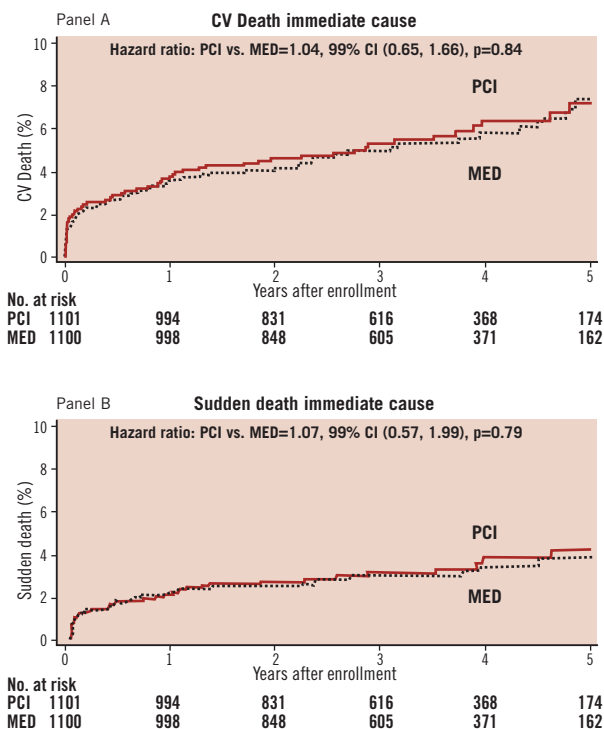

Figure 2. Life-table rates of death by treatment group for the outcomes cardiovascular death (Panel A) and sudden death (Panel B)

cerebrovascular disease {HR 2.28, p=0.002}, angina {HR 1.57, p=0.005}, history of heart failure {HR 2.11, p=0.004}, EF {HR 1.51 per 10% decrease, p<0.001}, days from MI to randomisation {HR 1.03 per day decrease, p=0.008}, and reduced GFR {HR 1.18, per 10 mL/min/1.73 m^2, p<0.001}.

Utilising Cox multivariable analysis, baseline variables independently associated with cardiovascular versus non-cardiovascular death were lower ejection fraction (p<0.01) and higher glomerular filtration rate (p<0.05). Variables independently associated with sudden cardiac death versus non-sudden death were lower ejection fraction (p<0.001) and younger age (p=0.001).

Cardiac rupture

Detailed characteristics of patients dying from cardiac rupture are shown in Table 3. Rupture of the free ventricular wall as an immediate cause of death occurred in four patients of the PCI group (on the same day in two patients, and on the 3rd and 8th day in two patients), and in two patients of the MED group. One of the latter had crossed over to PCI and suffered a PCI-related cardiac rupture. Thus, 83.3% of free wall rupture occurred within eight days of PCI (99% CI by exact binominal Clopper-Pearson test, 25.4% to 99.9%, p=0.22). Among these patients, one died of rupture after non-IRA PCI, and another after IRA and non-IRA PCI (patients #3 and 5, Table 3). Prior fibrinolytic therapy had been given to only 1/6 patients dying of cardiac rupture. Of the four patients with a reported final TIMI flow grade, TIMI 3 flow was achieved in only one.

Table 3. Characteristics of patients suffering from cardiac rupture.

Patient #	1	2	3	4	5	6
Treatment group	PCI	PCI	MED	PCI	PCI (IRA +non IRA)	MED
TIMI flow grade in IRA before PCI	0	0	1 (crossed over to non-IRA PCI)	0	0	0
TIMI flow grade in IRA after PCI	1	0	unknown	1	3	unknown
Time of death in relation to PCI	same day	same day	same day	3 days	8 days	no PCI
Adjudicated relation to procedure	PCI related	NOT PCI related	PCI related	NOT PCI related	NOT PCI related	NOT PCI related
Age	80	60	75	85	73	77
Gender	female	male	male	male	female	male
Thrombolysis	no	no	no	yes	no	no
Days from MI to randomisation	7	7	2	5	6	5
History of angina	yes	no	no	no	no	no
History of MI	no	no	no	no	no	no
History of cerebrovascular disease	yes	no	no	yes	no	no
History of peripheral vessel disease	no	no	no	yes	no	no
History of renal insufficiency	no	no	no	no	no	no
History of CHF	no	no	no	no	no	no
History of PCI	no	no	no	no	no	no
Cardiac risk factor - diabetes	no	no	no	no	no	no
Cardiac risk factor - hypertension	yes	no	yes	yes	no	yes
current cigarette smoker	no	yes	yes	no	no	no
Highest NYHA class	I	I	I	I	II	I
Highest Killip class	I	I	I	I	II	I
At randomisation						
Heart rate (baseline)	83	72	80	89	80	60
Systolic BP (baseline)	127	114	140	151	110	110
Diastolic BP (baseline)	86	76	80	89	80	70
Fasting glucose	112	103	119	121	125	94
Body mass index	23.1	26.2	31.2	27.6	24.9	28.7
Glomerular filtration rate	56.7	65	62.7	61.1	65.2	116
Ratio CKMB to ULN	na	na	31	3.8	9.5	na
Ratio CK to ULN	2.3	16.1	5.4	na	9.2	4.1
IRA	RCA	LAD	Circ	LAD	Circ	RCA
EF	NA	42	55	62	30	49
Collaterals	yes	yes	no	yes	yes	yes
Multivessel	no	yes	no	no	yes	no

Discussion

OAT challenged the late "open artery hypothesis" by demonstrating no reduction in death, reinfarction or heart failure during 5-year follow-up. These results confirm previous small studies[17], but came as some surprise because evidence for the superiority of primary PCI over medical therapy in the treatment of acute MI within 6-12 hours after onset of symptoms is overwhelming[18]. Because of the observed trend toward an excess risk of reinfarction in the PCI group[8], and evidence that suggests heart failure and sudden death may be reduced with PCI[19], a detailed descriptive analysis of causes of death was performed.

Immediate and primary causes of death were equally distributed between the assigned treatment groups, affecting 92 patients each.

Patients suffering death were generally older with comorbid conditions, e.g., diabetes and hypertension, and had lower LVEF as well as a higher likelihood of multivessel disease. Differences in medication use likely reflect poorer cardiovascular health status in the individuals suffering later death. Lower LVEF was a predictor of sudden and other cardiovascular death, while impaired kidney function predicted non-cardiovascular death. Older age predicted non-sudden death. If another component of the primary endpoint occurred prior to censoring for death, this event was likely to represent the cause of death. Predictors of death were overlapping with independent predictors of the composite outcome[20].

Contrary to hypothesis, when deaths were stratified by treatment assignment, no differences in cause or timing of death were evident.

According to MMCC review of deaths, before the uniform Academic Research Consortium criteria[21], only two stent thromboses occurred as cause of death, suggesting that this complication did not play a predominant role in the outcomes of OAT.

Few contemporary comparisons are available with early survivors of MI looking at long-term causes of death. In a landmark study describing the benefit of primary PCI over thrombolysis in acute MI who survived to 30 days, 5-year cumulative causes of death in the entire group were cardiac in 38 patients (9.6%), with a single case of cardiac rupture (0.2%), CHF in 14 cases (3.5%), and sudden death in 23 cases (5.8%)[22]. In a more recent study[23], the follow-up after AMI demonstrated a total of 158 cases of cardiac death in 1,009 patients (15.6%), with 68 patients dying within the first 30 days after AMI, seven of cardiac rupture (0.6%). The one-year cardiovascular death rate in OAT was 3.8%, with a rate of 0.9% mortality per year over the next four years. These data suggest a reduction in cardiac mortality with contemporary treatments. The observation in OAT that cardiovascular mortality decreased during the first year after MI and stabilised thereafter is in accord with published literature[23,24].

Cardiac rupture

Causes of death in patients who have survived at least 24 hours are skewed towards fewer acute arrhythmias and cardiogenic shock because OAT patients are, by definition, survivors of the early fatal arrhythmia and shock risk period. As a consequence, the relative proportion of cardiac rupture is higher in late presenters in general, and specifically in the OAT population, than would be expected in an overall STEMI population. Still, the frequency of free wall rupture (FWR) in the OAT PCI group is low (0,36% (4/1101) by "intention to treat" analysis, and 0,45% (5/1101) by "on treatment" analysis), and as such much lower than the reported risk of mechanical complications of MI treated with primary PCI (1.8%[25] and 2.2%[26]), or after combined primary PCI and thrombolysis (2.7 with optimal and 4.9% with failed PCI[27]). However, among the 41,021 patients in the Global Utilisation of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries (GUSTO-I) trial, ventricular septal rupture was confirmed by a retrospective review in 84 patients (0.2 percent)[28].

Although not statistically significant, it is interesting to note that five of six cases of FWR occurred in patients undergoing PCI (Table 3). Rupture of the free wall of the left ventricle is the second most common cause of death from acute MI in the coronary care unit, pump failure being the most common. Risk factors for rupture include older age, female sex, first infarction, moderate to large infarct size, an increased time delay from symptoms to treatment, hypertension, and absence of ventricular hypertrophy[29,30]. These characteristics apply to patients suffering rupture in OAT, with the exception that these were not more likely to be female (Table 3). Patients suffering rupture in OAT had documented collaterals to the IRA suggesting more long-standing ischaemia. The typical presentation of rupture is sudden cardiovascular collapse and death, but up to 30 percent of patients have a sub-acute form of rupture that may allow time for diagnosis and surgical intervention, with a greater possibility of survival[31-34]. Four of the cases of FWR in

OAT were adjudicated as not PCI-related but did occur within eight days of PCI. This is in keeping with current understanding of the timing of myocardial rupture, which has a bimodal time distribution, with approximately two-thirds occurring during the week after infarction, and one third within the first 24 hours[30,35]. In OAT, reperfusion timelines were covering a wide time window from >24 hours after the infarction to more than 200 hours later. It remains unknown when in the process if infarction the myocardium is most sensitive to rupture. Within a certain time window, successful PCI may decrease the area at risk and thus limit infarct expansion[36], however at the cost of an increased risk of intramyocardial haemorrhage. Fibrinolytic drugs are clearly associated with an increased risk of FWR[29]. The take-home message is that cardiac rupture has become a rare complication of contemporary myocardial infarction treatments, but may be precipitated by late PCI within days three and eight after the infarction.

Limitations

The number of patients suffering cardiac rupture is small, hence information and statistical analyses are limited.

We do not have information on the number of deaths in the initial 90 days after stopping clopidogrel[37].

Of 68 patients with sudden cardiac death, 20 may have met current criteria for ICD >30 days post MI, i.e., baseline EF<30% or NYHA Class >1 and EF < 35%[38]. In fact, seven of these patients suffered sudden cardiac death within 30 days of qualifying MI. Taken together, this calculation would translate into 13 deaths that may have been avoided in the current era. This figure marks an upper bound because some patients will have improved their EF by 30 days.

Although an independent MMCC adjudicated causes of death, attribution of the exact cause can be problematic in complex cases.

Conclusion

OAT provides insight into the causes of death in stable patients with persistent total occlusion of the IRA post MI, and the potential impact of delayed PCI. PCI of the culprit vessel does not affect the rate, cause or timing of death days to weeks after MI. The observation that the majority of cardiac ruptures occurred in patients undergoing PCI deserves further investigation.

Acknowledgements

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