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Scientific Research

Coronary Angiography in Patients with and without STEMI Following Out-of-Hospital Cardiac Arrest

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Abstract

Introduction: Little is known about discrepancies between patients who present with or without STEMI following out-of-hospital cardiac arrest (OHCA). Material and Methods: All patients with OHCA who were admitted to our hospital between January 1st 2008 and December 31st 2013 were classified according to their initial laboratory and electrocardiographic findings into STEMI, NSTEMI or no ACS. Results: Overall, 147 patients [32 STEMI (21.8%), 28 NSTEMI (19.0%) and 87 no ACS (59.2%)] were included with a mean age of 63.7 ± 13.3 years; there were 84 men (57.1%) and 63 (42.9%) women. Of these, 63 patients (51.7%) received coronary angiography [29 STEMI (90.6%), 9 NSTEMI (32.1%) and 38 no ACS (43.7%)] showing a high prevalence of coronary artery disease (CAD) [28 STEMI (96.6%), 9 NSTEMI (100.0%) and 26 no ACS (68.4%)] requiring percutaneous coronary intervention (PCI) in 52 cases [28 STEMI (96.6%), 8 NSTEMI (88.9%) and 16 no ACS (42.1%)]. Discussion: Coronary angiography immediately after hospital admission is feasible if all are prepared for potential further resuscitation efforts during cardiac catheterization. Primary focus on haemodynamic stabilisation may reduce the rates of coronary angiographies in patients following OHCA. Altogether, our data support the call for immediate coronary angiography in all patients following OHCA irrespective of their initial laboratory or electrocardiographic findings.

Keywords

Out-of-Hospital Cardiac Arrest, OHCA, Myocardial Infarction, STEMI, NSTEMI, Coronary Angiography, Resuscitation

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1. Introduction

There is a poor predictive value of clinical and electrocardiographic data, such as chest pain or ST-segment elevation (STEMI), for coronary-artery occlusion in patients following out-of-hospital cardiac arrest (OHCA). Therefore, the current Guidelines of the European Resuscitation Council emphasise early coronary angiography in all post-cardiac arrest patients who are suspected of having coronary artery disease irrespective of their electrocardiographic findings [1].

By now, few studies tried do further differentiate between patients with or without STEMI following OHCA. Pleskot *et al.* described ST-segment elevations as one of the strongest predictors for long-term survival following OHCA [2] and Anyfantakis *et al.* discussed whether the presence of ST elevation on admission might be used to triage OHCA patients to emergency angiography with a view to percutaneous coronary intervention (PCI) or not [3].

However, all these studies work on the basis of small patient's sizes: 26 patients with STEMI in the study of Pleskot *et al.* [2]; 27 patients with any type of myocardial infarction in the study of Anyfantakis *et al.* [3].

We, therefore, see the need for further studies on this subject to learn more about the discrepancies between patients who present with or without STEMI following OHCA.

2. Methods

2.1. Data Collection

All individuals with OHCA who were admitted to our hospital between January 1st 2008 and December 31st 2013 were identified by analysis of our central admission register. Individual patient data were collected from the patient's health records and anonymously stored on a central database. Statistical analysis was performed with the Statistical Package for Social Science (SPSS 22.0, IBM, Armonk, NY, USA). Continuous variables are expressed as the mean ± SD, comparisons of categorical variables among groups were conducted using Chi-square tests or student's t-test. Data collection and analysis was approved by the local ethical review committee.

2.2. Definition of Myocardial Infarction

ST elevation myocardial infarction (STEMI) was defined as new or presumably new significant ST-T changes in at least two corresponding leads or new Left Bundle Branch Block (LBBB) in combination with a rise and/or fall of cardiac biomarker values like troponin with at least one value above the 99th percentile of the upper reference limit during follow-up, but also in patients with cardiac death and presumably new ECG changes or new LBBB but death occurring before blood cardiac biomarker values would be increased [4].

Non-ST elevation myocardial infarction (NSTEMI) was defined as a rise and/or fall of cardiac biomarker values like troponin with at least one value above the 99th percentile of the upper reference limit but without significant ECG changes [4] [5].

If patients did not comply with one of the above described criteria, they were classified as without acute coronary syndrome (no ACS).

2.3. Treatment Strategy

In patients with suspected cardiac cause of death, coronary angiography was attempted as soon as possible, except in haemodynamically unstable patients where we primarily aimed at haemodynamic stabilisation.

In patients who received coronary angiography, catheterisation was performed according to standard techniques. If a recent coronary-artery stenosis was found, coronary angioplasty was attempted, unless the infarct-related artery was too small or the operator considered the procedure to be technically impossible. Standard resuscitative and stabilisation procedures were continued during the procedure if necessary.

3. Results

Altogether, 204 patients were admitted to our hospital between January 1st 2008 and December 31st 2013 following OHCA. Of these, 29 patients were excluded from further analysis due to absent 12-lead ECG as a consequence of incessant resuscitation efforts, 16 patients presented an ECG that could not be assessed according to ST segment elevation: four patients presented with continuous ventricular pacemaker stimulation, seven patients

with ventricular tachycardia and five patients with ventricular escape rhythm, and 12 patients had to be excluded due to missing or incomplete data. Clinical data of the remaining 147 patients are summarised in **Table 1**.

3.1. Patient's Characteristics

There were 84 men (57.1%) and 63 women (42.9%) with a mean age of 63.7 ± 13.3 years [range: 31 - 88 years]. Overall, there were 106 witnessed cardiac arrests (72.1%), lay resuscitation was attempted in 72 patients (49.0%) and 53 patients (36.1%) presented with an initial shockable rhythm.

Overall, 32 patients presented with an ST elevation myocardial infarction (STEMI), and 28 patients with a non-ST elevation myocardial infarction. Creatine kinase (CK) was 415.6 ± 733.0 U/l [range: 38 - 3898 U/l] and Creatine Kinase Muscle Brain (CK-MB) was 145.8 ± 153.4 (U/l) [range: 24 - 715]. Troponin was 3.5 ± 10.0 (ng/ml) [range: 0.0 - 50.0].

Cardiac catheterisation was performed in 76 patients (51.7%), 63 patients (42.9%) showed coronary artery disease (CAD), and 52 patients (35.4%) received percutaneous coronary intervention (PCI). Resuscitation during cardiac catheterisation was required in four patients (5.2%).

Also, 76 patients (51.7%) received mild therapeutic hypothermia (MTH, 33˚C for 24 hours, rewarming 0.3˚C /h) and 61 patients (41.5%) were discharged alive.

3.2. STEMI

Here, 32 patients presented with STEMI following OHCA; 16 of them (50.0%) were discharged alive, while 16

Table 1. Characteristics of those 147 patients following OHCA who could be included in our study.

	All Patients (n = 147)
Male	84 (57.1%)
Age (Years) [range]	63.7 ± 13.3 [31.0 - 88.0]
Witnessed cardiac arrest	106 (72.1%)
Lay resuscitation	72 (49.0%)
Initial shockable rhythm	53 (36.1%)
ST elevation myocardial infarction (STEMI)	32 (21.8%)
Non-ST elevation myocardial infarction (NSTEMI)	28 (19.0%)
Creatine kinase (CK) (U/l) [range]	415.6 ± 733.0 [38.0 - 3898.0]
Creatine kinase Muscle Brain (CK-MB) (U/l) [range]	145.8 ± 153.4 [24.0 - 715.0]
Troponin (ng/ml) [range]	3.5 ± 10.0 [0.0 - 50.0]
Coronary angiography	76 (51.7%)
Coronary artery disease (CAD)	63 (42.9%)
Percutaneous coronary intervention (PCI) RAMUS interventricularis anterior (RIVA) Ramus circumflexus (RCX) Right coronary artery (RCA) Multi vessel intervention (MVI)	52 (35.4%) 18 (12.2%) 7 (4.8%) 13 (8.8%) 14 (9.5%)
Resuscitation during cardiac catheterisation	4 (5.2%)
Mild therapeutic hypothermia (MTH)	76 (51.7%)
Discharged alive	61 (41.5%)

patients (50.0%) died in hospital. The only difference between these two groups was a higher Creatine kinase among patients who died (968.9 ± 1294.6 U/l vs. 280.8 ± 236.1 U/l) (p = 0.040). No differences could be seen in gender, age, rate of witnessed arrest, lay resuscitation, initial shockable rhythm, Creatine kinase Muscle Brain, Troponin at admission, cardiac catheterisation, coronary artery disease, percutaneous coronary intervention, necessity of resuscitation during cardiac catheterisation and application of mild therapeutic hypothermia (**Table 2**).

3.3. NSTEMI

Here, 28 patients following OHCA presented with NSTEMI; 7 of them (25.0%) survived until hospital discharge, while 21 died in hospital (75.0%). The only difference between patients with NSTEMI who survived until hospital discharge and patients who died in hospital was a higher rate of cardiac catheterisation among those patients who could be discharged alive (71.4% vs. 19.0%) (p = 0.010). No differences could be seen in gender, age, rate of witnessed arrest, lay resuscitation, initial shockable rhythm, Creatinine kinase, Creatinine kinase Muscle Brain, Troponin at admission, coronary artery disease, percutaneous coronary intervention, necessity of resuscitation during cardiac catheterisation and the application of mild therapeutic hypothermia (**Table 2**).

3.4. No ACS

Overall, 87 patients following OHCA presented with neither STEMI nor NSTEMI. In this group, 38 patients (43.7%) survived until hospital discharge, while 49 patients (56.3%) died in hospital. Patients who survived until

Table 2. Differences between patients who presented with STEMI, NSTEMI or without ACS following out-of-hospital cardiac-arrest.

	STEMI (n = 32)			NSTEMI (n = 28)			No ACS (n = 87)		
	Discharged Alive (n = 16)	Died in Hospital (n = 16)	p	Discharged Alive (n = 7)	Died in Hospital (n = 21)	p	Discharged Alive (n = 38)	Died in Hospital (n = 49)	p
Male	11 (68.8%)	9 (56.3%)	0.654	6 (85.7%)	12 (57.1%)	0.172	23 (60.5%)	23 (46.9%)	0.208
Age (Years)	59.8 ± 12.7	67.4 ± 13.6	0.108	67.3 ± 10.3	70.3 ± 13.1	0.589	63.5 ± 15.5	75.6 ± 12.1	<0.001
Witnessed cardiac arrest	14 (87.5%)	13 (81.3%)	0.831	6 (85.7%)	16 (76.2%)	0.695	29 (76.3%)	28 (57.1%)	0.175
Lay resuscitation	10 (62.5%)	8 (50.0%)	0.476	5 (71.4%)	7 (33.3%)	0.078	23 (60.5%)	19 (38.8%)	0.064
Initial shockable rhythm	11 (68.8%)	10 (62.5%)	0.690	5 (71.4%)	6 (28.6%)	0.055	15 (39.5%)	6 (12.2%)	0.002
Creatine kinase (CK) (U/l)	280.8 ± 236.1	986.9 ± 1294.6	0.040	690.1 ± 529.6	590.0 ± 971.5	0.798	171.4 ± 334.3	161.6 ± 197.5	0.871
Creatine kinase Muscle Brain (CK-MB) (U/l)	91.6 ± 35.9	210.6 ± 200.3	0.065	170.8 ± 75.5	160.9 ± 148.6	0.879	74.8 ± 51.8	77.2 ± 35.5	0.887
Troponin (ng/ml)	1.4 ± 2.2	7.2 ± 12.9	0.087	10.0 ± 15.9	5.0 ± 11.0	0.354	0.1 ± 0.1	0.1 ± 0.1	0.068
Coronary angiography	15 (93.8%)	14 (87.5%)		5 (71.4%)	4 (19.0%)		24 (63.2%)	14 (28.6%)	
<1 hour	8 (%)	6 (%)		3 (%)	1 (%)		8 (%)	5 (%)	
<4 hours	6 (%)	8 (%)	0.544	1 (%)	1 (%)	0.010	4 (%)	6 (%)	0.002
<24 hours	1 (%)	0 (0.0%)		0 (0.0%)	0 (0.0%)		5 (%)	2 (%)	
>24 hours	0 (0.0%)	0 (0.0%)		1 (%)	2 (%)		7 (%)	1 (%)	
Coronary artery disease (CAD)	14 (87.5%)	14 (87.5%)	1.000	5 (71.4%)	4 (19.0%)	0.171	14 (36.8%)	12 (24.5%)	0.163
Percutaneous coronary intervention (PCI)	14 (87.5%)	14 (87.5%)		4 (57.1%)	4 (19.0%)		8 (21.1%)	8 (16.3%)	
RAMUS interventricularis anterior (RIVA)	6 (37.5%)	4 (25.0%)		2 (28.6%)	0 (0.0%)		3 (7.9%)	3 (6.1%)	
RAMUS circumflexus (RCX)	1 (6.3%)	2 (12.5%)	1.000	1 (14.3%)	2 (9.5%)	0.053	1 (2.6%)	0 (0.0%)	0.552
Right coronary artery (RCA)	2 (12.5%)	6 (37.5%)		1 (14.3%)	0 (0.0%)		2 (5.3%)	2 (4.1%)	
Multi vessel intervention (MVI)	5 (31.3%)	2 (12.5%)		0 (0.0%)	2 (9.5%)		2 (5.3%)	3 (6.1%)	
Resuscitation during cardiac catheterization	1 (6.3%)	3 (18.8%)	0.285	0 (0.0%)	0 (0.0%)	-	0 (0.0%)	0 (0.0%)	-
Mild therapeutic hypothermia (MTH)	12 (75.0%)	9 (56.3%)	0.264	5 (71.4%)	9 (42.9%)	0.228	17 (44.7%)	24 (49.0%)	0.694

hospital discharge were significantly younger (63.5 ± 15.5 years vs. 75.6 ± 12.1 years) ($p < 0.001$), presented with an initial shockable rhythm more often (39.5% vs. 12.2%) and received cardiac catheterisation more often than those patients who died (63.2% vs. 28.6%) ($p = 0.002$). No differences could be seen in gender, rate of witnessed arrest, lay resuscitation, Creatinine kinase, Creatinine kinase Muscle Brain, Troponin at admission, coronary artery disease, percutaneous coronary intervention, necessity of resuscitation during cardiac catheterisation and the application of mild therapeutic hypothermia (**Table 2**).

4. Discussion

4.1. Patients with STEMI Following OHCA

STEMI following OHCA has been described as one of the strongest predictors for long-term survival, comparable to younger age or ventricular fibrillation as initial rhythm [2].

In our study that worked on this theme, there are four main findings.

First, the prevalence of relevant coronary artery stenosis is extremely high in patients with STEMI following OHCA, but almost all culprit lesions in these patients can be successfully treated by percutaneous coronary intervention [6] [7] (**Table 2** and **Figure 1**).

Second, higher Creatine kinase levels in patients who died during follow-up might underline the necessity of shortening the coronary no-flow time (**Table 2**). In our study, early coronary angiography within 60 minutes according to the guidelines for the treatment of patients with STEMI [4] could be achieved in almost half of all patients with STEMI (48.3%), irrespective the need for further airway management or haemodynamic stabilization in this special situation following OHCA.

Third, our results underline previous findings of a better prognosis of patients with STEMI following OHCA with survival rates of 50% or better [6] [8] [9]. Treating the cause of OHCA with coronary angiography and PCI obviously reduces the incidence of adverse events and decreases mortality during hospitalisation [10].

Fourth, enforcing early coronary angiography in patients with STEMI following OHCA is only feasible if all involved physicians and staff are prepared for further resuscitation efforts in the coronary laboratory (**Table 2** and **Figure 1**).

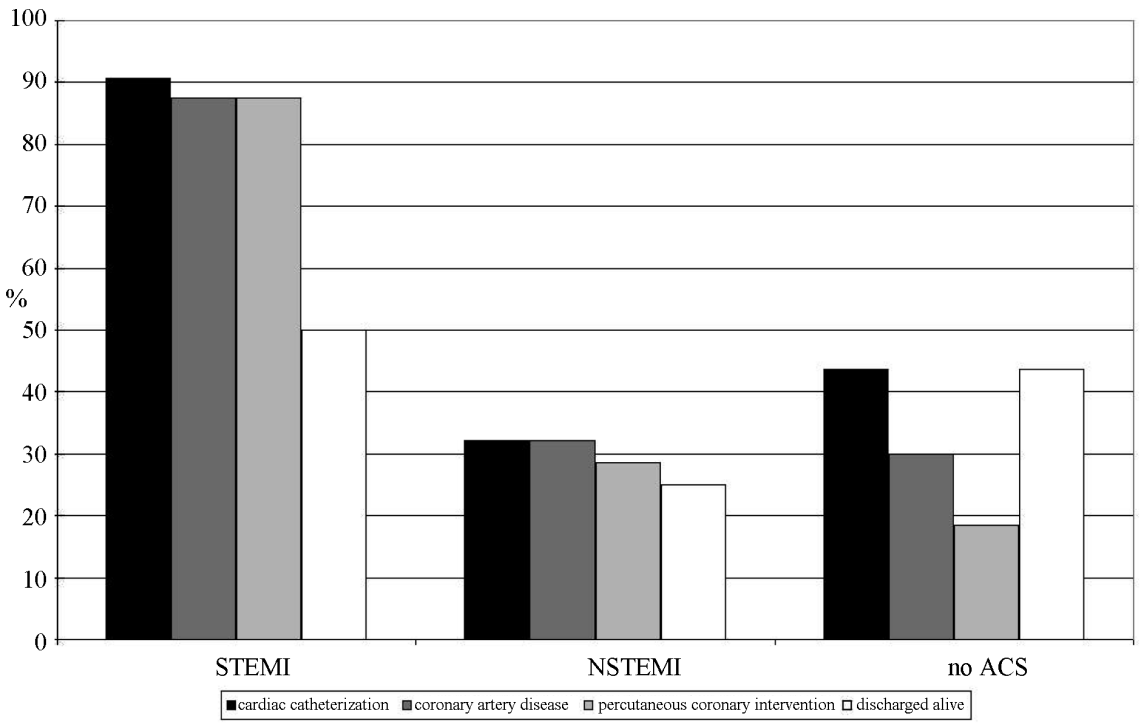

Figure 1. Survival rates in patients following OHCA depending on whether they initially present with STEMI, NSTEMI or no ACS.

4.2. Patients without STEMI following OHCA

In our study, also patients who presented with NSTEMI and no ACS following OHCA showed better survival rates if they received coronary angiography (**Table 2**). However, in these patients coronary angiography was attempted significantly later than in patients who presented with STEMI (**Table 2**). In our opinion, the main reason for this delay was a primarily focus on haemodynamic stabilisation in these patients as coronary angiography in these subgroups appeared less urgent than in patients who presented with STEMI; consequently, there were no further resuscitation attempts in the coronary laboratory in these groups.

However, in comparison with a primary focus on early PCI in patients with STEMI, a delay with primary focus on haemodynamic stabilisation led to much lower rates of coronary angiography in patients with NSTEMI (32.1%) or no ACS (43.7%).

Of course, this is a single centre study and the rates of early coronary catheterisation and PCI vary enormously among hospitals [11]. However, our data are in line with other investigations that repetitively described rates of less than 50% for early coronary angiography if patients presented without STEMI following OHCA [12]-[14]. Following our findings, a too cautious proceeding in patients without STEMI might be one of the main reasons why there are discrepant findings and opinions about the benefit of early PCI in these patients [3] [8] [15]-[18].

However, the probability of patients who do not receive early coronary angiography on the first day after hospital admission receiving later coronary angiography during the same hospital stay was extremely low in our data (7.5%) (**Table 2**). There might be several reasons for such a low rate of later coronary angiographies during follow-up: infections, sepsis, respiratory insufficiency, kidney injury, patient's decision, or maybe an obvious extra-cardiac cause of OHCA; however, the enormous discrepancies between our three groups underline the importance of the initial decision to undergo early coronary angiography or not.

Unfortunately, there are still hardly any objective data that can be used for the initial decision at admission. To date, no biomarker has been shown to correctly predict culprit coronary occlusion in OHCA patients [19] and also the dosage of cardiac troponin I at admission could not help in the decision of early coronary angiogram previously [20]. Also in our study, troponin and Creatine kinase at admission did not differ between patients without STEMI who survived and those who died during follow-up (**Table 2**). Only in patients with STEMI was higher Creatine kinase as an indirect marker of myocardial damage associated with worse survival rates (**Table 2**).

Likewise, also ST-segment analysis might have a good positive predictive value but a low negative predictive value in diagnosing the presence of acute or presumed recent coronary artery lesions [17]. In our study, 88.9% of the patients with NSTEMI and 42.1% of the patients with no ACS who received coronary angiography also received percutaneous coronary intervention, underlining the high prevalence of relevant coronary artery stenosis in patients following OHCA, irrespective of the initial electrocardiographic or laboratory findings (**Table 2**).

Therefore, our results support previous statements that electrocardiographic findings after OHCA should not be considered as strict selection criteria for performing emergent coronary angiography in patients resuscitated from OHCA without an obvious extra-cardiac cause [8]. Even in the absence of ST-segment elevation on post-ROSC ECG, acute culprit coronary lesions may be present and considered the trigger of cardiac arrest.

4.3. Limitations

Our data are the result of a single centre study; rates of early coronary catheterisation and coronary intervention vary enormously between hospitals [11]. However, our data are in line with several previous investigations [12]-[14] and findings of better survival rates in younger patients and those with an initial shockable rhythm (**Table 2**) also affirm a representative patient population.

5. Conclusions

The observation of better survival rates in patients with STEMI and immediate PCI underlines the benefit of an immediate treatment of the cause of death in these patients. However, considering the high prevalence of coronary artery disease in patients who present without any electrocardiographic or laboratory signs of myocardial ischaemia, percutaneous coronary intervention may be a necessary causal treatment in many more cases.

All authors declare no conflicts of interest.

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