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par

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Comparaison des patients ayant fait un arrêt cardio-respiratoire extrahospitalier avec ou sans sus décalage du segment ST et place de la coronarographie chez ces patients :

une étude monocentrique rétrospective au CHU de Tours

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SERMENT D'HIPPOCRATE

En présence des enseignants et enseignantes
de cette Faculté,
de mes chers condisciples
et selon la tradition d'Hippocrate,
je promets et je jure d'être fidèle aux lois de l'honneur
et de la probité dans l'exercice de la Médecine.

Je donnerai mes soins gratuits aux indigents,
et n'exigerai jamais un salaire au-dessus de mon travail.

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les secrets qui me seront confiés et mon état ne servira pas
à corrompre les mœurs ni à favoriser le crime.

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l'instruction que j'ai reçue de leurs parents.

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Résumé

Comparaison des patients ayant fait un arrêt cardio-respiratoire extrahospitalier avec ou sans sus décalage du segment ST et place de la coronarographie chez ces patients : une étude monocentrique rétrospective au CHU de Tours

Objectif

L'infarctus du myocarde est une des principales causes d'arrêt cardiaque extrahospitalier (ACR-EH). Cependant, la place de la coronarographie en urgence, bien établie dans les infarctus du myocarde avec sus-décalage du segment ST (ACR-EH-ST+), reste incertaine dans certaines situations et notamment, celles des ACR-EH sans sus-décalage du segment ST (ACR-EH-ST-).

Les dernières recommandations de l'ESC et de l'ACC/AHA ne définissent pas clairement le délai optimal de la coronarographie dans les ACR-EH-ST-. Ces dernières années, trois essais randomisés (COACT, 2019 / TOMAHAWK, 2021 / EMERGE, 2022) ont étudié la place de la coronarographie (en urgence vs différée) chez les patients ACR-EH-ST-, mais n'ont pas réussi à montrer un bénéfice sur la mortalité de la coronarographie en urgence.

Matériel et méthode

Notre étude rétrospective monocentrique au CHU de Tours, sur les dix dernières années, de janvier 2012 à décembre 2022, étudie l'impact clinique de la réalisation immédiate de la coronarographie chez les patients ayant fait un ACR-EH avec ou sans ST+ à l'électrocardiogramme, ainsi que l'évolution de nos pratiques.

Notre étude a inclus tous les patients consécutifs pris en charge pour ACR-EH et adressés directement en salle de coronarographie après récupération d'une activité cardiaque. Ils ont été répartis en deux sous-groupes : ACR-EH-ST+ et ACR-EH-ST-. Notre principal critère de jugement était la mortalité toute cause à 30 jours. Les critères de jugement secondaires étaient le score de Glasgow à l'admission en réanimation, la durée de l'hospitalisation en réanimation, la durée d'intubation, la durée du support inotrope et les critères secondaires de sécurité étaient un saignement majeur, un AVC, des troubles du rythme ventriculaire et une insuffisance rénale aigue nécessitant un recours à la dialyse.

Résultats

237 patients pris en charge pour ACR-EH ont été inclus, dont 124 ACR-EH-ST+ et 113 ACR-EH-ST-. Les patients ACR-EH-ST- étaient significativement plus âgés et plus à risque, avec plus souvent des antécédents de maladie cardiovasculaire, que les patients ACR-EH-ST+. A 30 jours, 53 (43%) patients du groupe ACR-EH-ST+ et 64 (57%) patients du groupe ACR-EH-ST- étaient décédés ($p<0,05$). La réalisation en urgence de la coronarographie post ACR-EH retarde l'admission en unité de réanimation et la mise en place des mesures de neuroprotection (avec un temps moyen de transfert de 100 minutes compte tenu de la distance entre la salle de coronarographie et l'unité de réanimation dans notre CHU), ce qui pourrait expliquer en partie la surmortalité du groupe ACR-EH-ST-, chez qui seulement 21% de lésions coronaires coupables ont été retrouvées. En analyse multivariée, seuls l'âge et l'absence de rythme choquable restaient des prédicteurs de mortalité.

Conclusion

La prise en charge des ACR-EH suspects d'une étiologie cardiaque dans notre centre est cohérente avec les résultats des dernières recommandations et études. Le recours à la coronarographie en urgence semble plus bénéfique chez les patients avec sus décalage post ressuscitation. Notre étude n'a pas réussi à identifier de nouveaux facteurs prédictifs permettant de mieux sélectionner les patients avec un ST- post ACR-EH pouvant bénéficier de la coronarographie en urgence avant l'admission en réanimation.

Mots clés

Arrêt cardiaque extrahospitalier, sous-décalage, sus-décalage, coronarographie, maladie coronaire, lésion coronaire coupable, neuroprotection, mortalité toute cause

Abstract

Comparison of emergency coronary angiography in ST elevation and non-ST elevation out-of-hospital cardiac arrest patients: a monocenter retrospective study

Background

Myocardial infarction is one of the major cause of out-of-hospital cardiac arrest (OHCA). However, the place of immediate coronary angiography (CAG), clearly indicated in ST-segment elevation myocardial infarction (STEMI), remains uncertain with other clinical presentations such as non ST elevation (NSTE), acute coronary syndrome these last years. ESC and ACC/AHA guidelines do not clearly define the timing of CAG in OHCA without STEMI. Three recent prospective randomized trials COACT (2019), TOMAHAWK (2021) and EMERGE (2022) did not prove a benefit of an immediate CAG over a delayed CAG with respect to 30-day risk of death from any cause in NSTE-OHCA patients.

Objective

Our retrospective study seeks to assess the clinical impact of immediate coronary angiography in OHCA patients, with or without ST-segment elevation in University hospital of Tours during the last decade, in order to compare our practice and results to these recent randomized studies.

Materials and Methods

This monocentric retrospective observational study was based on a cohort of 237 patients, who were referred to the catheterization laboratory for an immediate coronary angiography after an out-of-hospital cardiac arrest, from January 2012 to December 2022, in the university hospital center of Tours, in France. A subgroup analysis was performed according to post resuscitation ECG (STE-OHCA group and NSTE-OHCA group). Our main end point was death from any cause at 30 days. Our secondary efficacy end points were neurological deficit (Glasgow Coma Scale at the discharge of the ICU), length of stay in the ICU, length of mechanical ventilation, length of inotropic support, and our secondary safety end points were severe bleeding, stroke, ventricular tachycardia, and acute kidney failure leading to renal-replacement therapy.

Results

At 30 days, 53 of 124 patients (43%) in the STE-OHCA group and 64 of 113 patients (57%) in the NSTE-OHCA group had died (p<0,05). Immediate CAG in OHCA with cardiac cause (STE or No STE group) in our center postpones ICU admission and neuroprotection management (median time of transfer around 100 minutes), which could explain the higher mortality in NSTE-OHCA group, as coronary culprit lesion was only identified in 21% of these patients. Multivariate analysis showed that both age and absence of shockable first monitored rhythm after resuscitation remained the only significant predictors of mortality at 30 days.

Conclusion

Management of OHCA patients with cardiac cause (ST elevation or Non ST elevation) in our center is consistent with observed shift of scientific societies recommendations during the last decade, as we saw an evolution in the profile of patient undergoing an immediate CAG after OHCA (exclusively the STE-OHCA). Our retrospective study failed at identifying predictive factors of a benefit of early CAG in NSTE-OHCA patients.

Key words

Out-of-hospital cardiac arrest, Non-ST elevation, ST elevation, coronary angiography, coronary artery disease, culprit lesion, neuroprotection, death from any cause

Abbreviations

BBB: Bundle Branch Block

BMI : Body Mass Index

CAG: Coronary Angiography

CABG : Coronary Artery Bypass Grafting

Cath Lab : Catheterization Laboratory

CPC Scale : Cerebral Performance Categories Scale

CPR : CardioPulmonary Resuscitation

ECG : Electrocardiogram

EEC : External Electric Shock

GCS : Glasgow Coma Scale

ICU: Intensive Care Unit

LF: Low Flow

NF: No Flow

NSE : Neuron Specific Enolase

NSTE: No ST Segment Elevation

NSTE-OHCA: No ST Segment Elevation – Out of Hospital Cardiac Arrest

OHCA: Out-of-Hospital Cardiac Arrest

PCI : Percutaneous Coronary Intervention

STE: ST Segment Elevation

STE-OHCA: ST Segment Elevation – Out of Hospital Cardiac Arrest

STEMI : ST- Elevation Myocardial Infarction

VT : Ventricular Tachycardia

Introduction

Out-of-hospital cardiac arrest (OHCA) is a frequent cause of death worldwide and thus a major public health issue. More than 40,000 patients suffer from OHCA in France every year (i.e. one OHCA every 10 minutes [1]), with an estimated 300,000 OHCA per year in Europe [2], and almost 350,000 OHCA per year in the United States of America [3].

The prognosis of OHCA is poor and frequently associated with a low survival rate. Indeed, despite the advances in cardiopulmonary resuscitation (CPR) and the progression in emergency and intensive care, mortality remains high. Return to spontaneous circulation after cardiac arrest only occurs in 30% of OHCA, and few patients arrive alive at the hospital (22%). In-hospital survival is estimated at 8%, with subsequent one-month survival rate of 10% and a one-year survival rate of 7,7% [2,4].

Cardiac cause is found in 65% of all cardiac arrests[5]. In most cases (80%), acute coronary syndrome or chronic coronary syndrome is found to be responsible of the cardiac arrest, before non-ischemic related ventricular arrythmia[6]. The place of coronary angiography (CAG) is critical and plays a central role in patient care, as early revascularization improves survival rate and prognosis in patients with severe acute myocardial ischemia[7]. However, even if the timing of the CAG in OHCA with ST segment elevation (STE-OHCA) is clear[8], it is less evident for OHCA without STE (NSTE-OHCA)[9–12]. Even though early revascularization has proved its efficacy by reducing mortality and improving cardiac prognosis, the place of intensive care management of cardiorespiratory arrest also plays an important role[13], especially cerebral neuroprotection (survival rate without neurological deficit is improved by early neuroprotection care).

Thus, transferring OHCA patient to a cath lab for an emergency CAG is likely to delay intensive care unit (ICU) admission and initiation of neuroprotection strategies. ESC and ACC/AHA guidelines[14–16] do not clearly define the timing of CAG in NSTE-OHCA patients management.

Since 2018, several randomized controlled trials investigating the timing of CAG in patients with NSTE-OHCA were published[17–19]. Among them, three large randomized prospective clinical trials, COACT (2019)[20], TOMAHAWK (2021)[21] and EMERGE (2022)[22] specifically investigated the question of immediate versus delayed CAG in NSTE-OHCA patients.

These studies did not show a significant benefit of immediate vs delayed CAG in NSTE-OHCA patients. There was no statistical difference on the overall survival at 30 or 90 days. These information are important for pre hospital health care professional, emergency physician and interventional cardiologist in charge of triaging and managing NSTE-OHCA patients. The decision to transfer the patients to the cath lab before the ICU may impact its prognosis, especially in a situation where both facilities are not at the same hospital.

As this is the case in our university hospital, we performed a retrospective clinical study on resuscitated OHCA patients referred for emergency CAG to our cath lab in the last 10 years.

Materials and Methods

TRIAL DESIGN AND OVERSIGHT

The study was designed as a monocentric retrospective observational study. The study enrolled a cohort of all consecutive patients who were referred to the catheterization laboratory for an immediate coronary angiogram after a resuscitated OHCA without exclusion criteria, from January 2012 to December 2022, in the university hospital center of Tours, in France.

PATIENTS

Patients were eligible for the study if they had been referred to the cath lab for a fast track CAG after an out of hospital cardiac arrest followed by the recuperation of spontaneous rhythm. Decision to perform an immediate CAG was taken jointly by the cardiologist, the emergency and the intensive care physicians.

Patients were eligible only if post CAG hospitalization was in one of the different ICU of the university hospital of Tours. Exclusion criteria were in-hospital cardiac arrest (in operating room, in emergency department…), no hospitalization in ICU after the CAG, death before the arrival in ICU, or obvious non-coronary cause of the cardiac arrest (sepsis…).

Data were gathered from our (CardioReport™, MediReport, cath lab data base) and from our electronic clinical record manager (DPP, Cerner, Millenium®).

380 patients were recorded on our cath lab database for "cardiac arrest" from January 2012 to December 2022. 237 (62%) patients were enrolled following our inclusion criteria (OHCA with a suspected coronary cause leading to immediate CAG) and 143 (38%) were excluded (48 (13%) because of in hospital cardiac arrest, 38 (10%) were hospitalized in ICU in another hospital of Loire Valley, 15 (4%) did not have cardiac arrest, 13 (3%) died before the admission in ICU, 9 (2%) had a delayed CAG and 20 (5%) for other cause) (Figure 1).

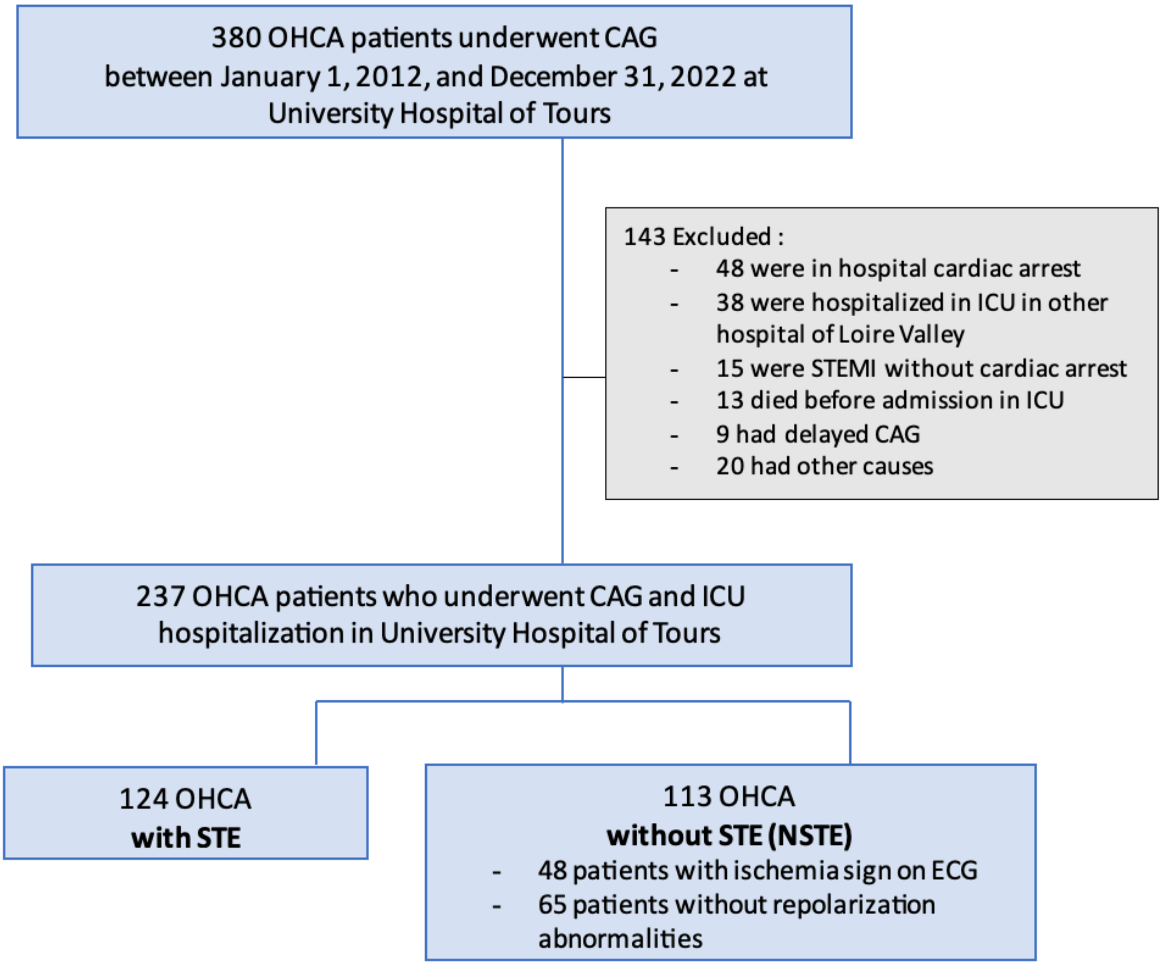

Figure 1. Flow Chart of the study

PATIENT CHARACTERISTICS AND TREATMENT

The main patient characteristics studied were age, sex, body mass Index (BMI), cardiovascular risk factors, previous coronary or peripheral artery disease, previous percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG), previous stroke, heart surgery or chronic kidney or respiratory failure.

The circumstances of the cardiac arrest were collected (arrest witnessed, bystander cardiopulmonary resuscitation, shockable first monitored rhythm, median time from arrest to basic life support (no flow), median time from arrest to return of spontaneous circulation (low flow)).

Post resuscitation first ECG analysis was retrieved from the physician's interpretation recorded in the medical report (sign of ischemia, rhythm, repolarization abnormality). Following our local protocol, an echocardiography was realized, if possible, at the arrival of the patient in the cath lab. The CAG retrospectively collected data were the median time from arrest to CAG, the median time of the CAG, the catheterization access, the severity of coronary artery disease, the identification of the culprit lesion (defined as a severe artery stenosis at least of 70% or occlusion), the revascularization treatment, and the median amount of contrast dye. In patients with multivessel disease, the choice of revascularization strategy was at the discretion and local protocol of the individual cath lab operator. In ICU, Glasgow Coma scale, temperature and biological laboratory values were collected from electronic clinical report for each patient. Post resuscitation care was in line with international and European resuscitation guidelines. Targeted temperature management was initiated as soon as possible and was performed in accordance with local protocol of the different ICU of the University hospital of Tours (medical ICU, surgical ICU, cardiac surgical ICU, URTC ICU). Prevention and control of secondary cerebral injury of systemic origin were managed according to guidelines. In patient with persistent coma, the life sustaining treatment was based on local practice.

PRIMARY AND SECONDARY END POINTS

We first studied the cohort and then divided it into two groups based on post-resuscitation ECG analysis. The first group included all the patients with ST elevation (STE-OHCA group) and the second group, all patients without ST elevation (NSTE-OHCA group). We compared each groups. Our main end point was death from any cause at 30 days. Our secondary efficacy end points were neurological deficit (Glasgow Coma Scale at the discharge of the ICU), length of hospitalization in ICU, length of mechanical ventilation, length of inotropic support. Our secondary safety end points were severe bleeding, stroke, ventricular tachycardia, acute kidney failure leading to renal-replacement therapy. We also defined biological secondary endpoints by using a median creatinine peak level, a median troponin ratio and median NSE level usually measured at 2-3 days of ICU hospitalization. The troponin ratio was calculated by the troponin level divided by the standardized level norm, as the bioassay of troponin changed during the years (troponin I and then troponin T hs), as well as the standardized norms (<0,07 for the troponin I and <14 for troponin T hs).

STATISTICAL ANALYSIS

Categorical variables are expressed as percentages. Quantitative variables are expressed as median with IQR. We used Chi2 Test to compare categorical variables and Student T test to compare quantitative variables. Logistic regression was used for univariate analysis and for multivariate analysis, on R software (R Core Team, 2020). A difference was considered significant, if the p value was less than 0,05 ($p<0,05$).

Results

From January 2012 to December 2022, we retrospectively enrolled 237 patients who had been successfully resuscitated after OHCA and were immediately referred to the cath lab of University hospital of Tours for emergency CAG.

124 patients (52%) had a ST-segment elevation on ECG (STE-OHCA group) and 113 patients (48%) did not have a ST-segment elevation on ECG after resuscitation (NSTE-OHCA group).

PATIENT CHARACTERISTICS

The baseline characteristics are shown in Table 1. Median age was 63 years, with a majority of men (76%) with a high cardiovascular risk.

Table 1 . Baseline Characteristics of the Cohort

	COHORT (N= 237)
Median age (IQR) – year	63 (51-71)
Male sex -no. (%)	180 (76)
Median BMI (IQR) – kg/m2	27 (24-31)
Cardiovascular risk – no. (%)	
• Diabetes mellitus	45 (19)
• Hypertension	114 (48)
• Current smoker	79 (33)
• Dyslipidemia	76 (32)
Previous coronary artery disease – no. (%)	35 (15)
Previous peripheral artery disease – no. (%)	14 (6)
Previous PCI – no. (%)	26 (11)
Previous CABG – no. (%)	7 (3)
Previous stroke – no. (%)	9 (4)
Previous heart surgery – no. (%)	8 (3)
Chronic kidney failure – no. (%)	5 (2)
Chronic respiratory failure – no. (%)	1 (0,4)

In our subgroups analysis, baseline characteristics were quite similar between groups except for age and past history of coronary artery disease. Patients of the NSTE-OHCA group were significantly older (65 [55-76] versus 58 [50-69], p=0,01). There was also a statistically significant difference between STE-OHCA group and NSTE-OHCA group regarding the proportion of previous coronary artery disease (10% in STE-OHCA group vs 20% in NSTE-OHCA group, p<0,05). Thus, we observed that the number of previous PCI was higher as well in NSTE-OHCA group (Table 2). This is consistent with a higher proportion of patients receiving baseline aspirin medication in NSTE-OHCA group than in STE-OHCA group (p<0,05) (Table 11).

Table 2. Comparison of baseline Characteristics of the Patients			
	STE-OHCA (N= 124)	NSTE-OHCA (N=113)	*p-value*
Median age (IQR) – year	58 (50-69)	65 (55-76)	**0,01**
Male sex – no. (%)	94 (76)	83 (73)	0,92
Median BMI (IQR) – kg/m2	27 (24-31)	27 (23-30)	0,36
Cardiovascular risk – no. (%)			
• Diabetes mellitus	18 (15)	26 (2)	0,10
• Hypertension	55 (45)	58 (51)	0,70
• Current smoker	45 (37)	33 (29)	0,23
• Dyslipidemia	33 (27)	42 (37)	0,09
Previous coronary artery disease – no. (%)	12 (10)	23 (20)	**< 0,05**
Previous peripheral artery disease – no. (%)	5 (4)	9 (8)	0,32
Previous PCI – no. (%)	8 (7)	18 (16)	**< 0,05**
Previous CABG – no. (%)	2 (2)	5 (4)	0,26
Previous stroke – no. (%)	7 (6)	2 (2)	0,18
Previous heart surgery – no. (%)	4 (3)	4 (4)	1,00
Chronic kidney failure – no. (%)	0 (0)	5 (4)	**< 0,05**
Chronic respiratory failure – no. (%)	0 (0)	1 (1)	0,48

CARDIAC ARREST CIRCUMSTANCES

In our cohort, a witness was present in 84% of OHCA. 73% of patients benefited from cardiopulmonary resuscitation initiated by a bystander and 69% had a shockable first monitored rhythm. No Flow (NF) and Low Flow (LF) median durations were respectively 5 minutes and 20 minutes (Table 3).

Table 3. Cardiac arrest circumstances in the cohort	
	COHORT (N= 237)
Arrest witnessed – no. (%)	200 (84)
Bystander CPR – no. (%)	174 (73)
Shockable first monitored rhythm – no. (%)	163 (69)
Median time from • arrest to basic life support **(No Flow)** (IQR) – min • arrest to return of spontaneous circulation **(Low Flow)** (IQR) – min	 5 (1-10) 20 (13-27)

In the STE-OHCA and NSTE-OHCA groups, there were no significant differences regarding the presence of a witness or the initiation of cardiopulmonary resuscitation by a bystander. The two significant differences were a higher frequency of shockable first monitored rhythm in STE-OHCA group (77% vs 58%, p<0,001) and a shorter median NF duration (3 min vs 5 min, p<0,05) (Table 4).

Table 4. Comparison of cardiac arrest circumstances between STE-OHCA and NSTE-OHCA			
	STE-OHCA (N= 124)	NSTE-OHCA (N=113)	*p-value*
Arrest witnessed – no. (%)	104 (84)	92 (81)	1,00
Bystander CPR – no. (%)	95 (77)	75 (66)	0,19
Shockable first monitored rhythm – no. (%)	95 (77)	65 (58)	**< 0,001**
Median time from • arrest to basic life support **(No Flow)** (IQR) – min • arrest to return of spontaneous circulation **(Low Flow)** (IQR) – min	 3 (0-5) 20 (12-28)	 5 (1-10) 20 (14-30)	 **< 0,05** 0,62

ECG

In our cohort, post resuscitation ECG analysis shows a majority of narrow QRS (69%). Repolarization abnormalities were localized in anterior and inferior leads (18% each) (Figure 5. in Supplementary data).

Post resuscitation ECG analysis shows that ischemia signs were more frequently localized in anterior (29%) and inferior (31%) leads with narrow QRS (85%) in the STE-OHCA group than in NSTE-OHCA group, where narrow QRS were significantly less frequent (51%) and lateral localization ischemia was predominant (12%) (Figure 5. in Supplementary data).

TREATMENTS

Details about procedures, severity of coronary artery disease and treatments are provided in Table 5.

Table 5. Procedures, treatments, and characteristics of coronary artery disease	
	COHORT (N= 237)
Median time from arrest to coronary angiography (IQR) – min	135 (107-170)
Median time of coronary angiography (IQR) – min	19 (11-33)
Catheterization access – no. (%)	
• Radial	192 (81)
• Femoral	41 (17)
Severity of coronary artery disease – no. (%)	
• No significant disease	75 (32)
• One-vessel disease	159 (67)
• Two-vessel disease	60 (25)
• Three-vessel disease	54 (23)
Culprit lesion identified – no. (%)	115 (49)
Revascularization treatment – no. (%)	
• PCI performed	101 (43)
• CABG	1 (0,4)
• Pharmacologic or conservative treatment	59 (25)
Median amount of contrast dye (IQR) – mL	145 (52-120)

The median time from arrest to coronary angiography was similar in both groups: 135 minutes in STE-OHCA group and 130 min in NSTE-OHCA group (p=0,12). Radial access was routinely chosen as a first line access in both groups (p=0,77). The median time of CAG was longer in STE-OHCA group than in NSTE-OHCA group (p<0,01). Culprit lesion was identified in 49% of all OHCA. However, we observed a significant difference between STE-OHCA group and NSTE-OHCA group. A culprit lesion was identified in 89 patients of STE-OHCA group (72%) and only in 24 patients of NSTE-OHCA group (21%) (p<0,001). Bitroncular and tritroncular lesions rate were not statistically different between groups.

The revascularization by PCI rate was 43% in the entire cohort (whereas it was 49% of patients with a culprit lesion identified). We observed that in STE-OHCA group, treatment of the identified culprit lesion by PCI was made as a first choice in 66% of patients vs 14% in the NSTE-OHCA group (p<0,001). On the contrary, in the NSTE-OHCA group, medical treatment was prioritized in 35% of patients vs 15% in the STE-OHCA group. Surgery revascularization by CABG was realized for one patient in STE-OHCA group. The amount of contrast dye was significantly higher in STE-OHCA than in NSTE-OHCA (p<0,001), certainly due to a longer time of CAG and higher rate of revascularization by PCI (Table 6).

Table 6. Procedures, treatments, and characteristics of coronary artery disease

	STE-OHCA (N= 124)	NSTE-OHCA (N=113)	*p-value*
Median time from arrest to coronary angiography (IQR) – min	135 (110-174)	130 (100-161)	0,12
Median time of coronary angiography (IQR) – min	25 (14-39)	15 (10-20)	**< 0,01**
Catheterization access – no. (%)			
• Radial	98 (79)	90 (80)	0,77
• Femoral	23 (19)	18 (16)	0,67
Severity of coronary artery disease – no. (%)			
• No significant disease	23 (19)	50 (44)	0,06
• One-vessel disease	39 (32)	22 (20)	0,10
• Two-vessel disease	32 (26)	22 (20)	0,54
• Three-vessel disease	29 (23)	15 (13)	0,30
Culprit lesion identified – no. (%)	89 (72)	24 (21)	**< 0,001**
Revascularization treatment – no. (%)			
• PCI performed	82 (66)	16 (14)	**< 0,001**
• CABG	1 (1)	0 (0)	1,00
• Pharmacologic or conservative treatment	18 (15)	39 (35)	**< 0,001**
Median amount of contrast dye (IQR) – mL	92 (61-132)	63 (50-99)	**< 0,001**

ICU

Intensive care management were similar in the two groups with no statistically significant differences (Table 8). In average, 100 minutes were necessary to transfer patients from the cath lab to the ICU (the main ICU being in a different hospital at 8.6 km distance) for the STE-OHCA group and 90 minutes for the NSTE-OHCA group (p=0,49).

Table 7. Baseline characteristics at admission in ICU	
	COHORT (N= 237)
Median time from cath lab to ICU transfer (IQR) – min	100 (77-123)
Median score range of Glasgow Coma Scale at admission in ICU (IQR)	3 (3-3)
Median blood pressure (IQR) – mmHg	
• Systolic	121 (101-140)
• Diastolic	71 (60-85)
• PAM	87 (75-101)
Inotropic support – no. (%)	120 (51)
Median temperature (IQR) – °Celsius	35,4 (34,5-36,1)
Median left ventricular ejection fraction (IQR) – %	40 (30-50)
Baseline laboratory values	
• pH	7,31 (7,23-7,37)
• Median lactic acid (IQR) – mmol/liter	2,5 (1,5-4,2)
• Median partial pressure of O_2 (IQR) – kPa	118 (83-193)
• Median partial pressure of CO_2 (IQR) – kPa	41 (38-46)
• Median creatinine (IQR) – µmol/liter	98 (81-124)
• Median troponin ratio (IQR)	43 (10-119)
• Median hemoglobin (IQR) – (g/dl)	13,7 (12,4-14,9)
Hypothermia protection – no. (%)	118 (50)
Pupillary reflex presence – no. (%)	138 (58)

Median temperature at the arrival in ICU was 35,5° (STE-OHCA) and 35,3° (NSTE-OHCA group), and hypothermia protection was initiated in 47% in STE-OHCA group vs 52% in NSTE-OHCA group. The median length of ICU hospitalization, of mechanical ventilation or of inotropic support were similar and not statistically significantly different (Table 8).

Table 8. Baseline characteristics at admission in ICU			
	STE-OHCA (N= 124)	NSTE-OHCA (N=113)	*p-value*
Median time from cath lab to ICU transfer (IQR) – min	100 (82-127)	90 (70-120)	0,49
Median score range of Glasgow coma scale at admission in ICU (IQR)	3 (3-3)	3 (3-3)	0,42
Median blood pressure (IQR) – mmHg			
• Systolic	121 (103-136)	121 (103-140)	0,59
• Diastolic	72 (64-85)	68 (60-84)	0,31
• PAM	89 (78-100)	87 (75-103)	0,96
Inotropic support – no. (%)	66 (53)	52 (46)	0,41
Median temperature (°Celsius) (IQR)	35,5 (34,6-36,2)	35,3 (34,4-36)	0,97
Median left ventricular ejection fraction (IQR) – %	40 (30-50)	45 (30-60)	0,35
Baseline laboratory values			
• pH	7,32 (7,25-7,37)	7,29 (7,20-7,36)	0,21
• Median lactic acid (IQR) – mmol/liter	2,4 (1,4-3,9)	2,7 (1,8-4,3)	0,32
• Median partial pressure of O_2(IQR) – kPa	111 (82-165)	140 (93-227)	**< 0,05**
• Median partial pressure of CO_2 (IQR) – kPa	41 (37-47)	43 (38-46)	0,74
• Median creatinine (IQR) – µmol/liter	94 (78-114)	105 (90-134)	**< 0,001**
• Median troponin ratio (IQR)	79 (25-181)	14 (5-53)	**< 0,001**
• Median hemoglobin (IQR) – (g/dl)	13,6 (12,5-15,1)	13,8 (12,2-14,8)	0,22
Hypothermia protection – no. (%)	58 (47)	59 (52)	0,33
Pupillary reflex presence – no. (%)	71 (57)	66 (58)	0,45

PRIMARY AND SECONDARY END POINTS

Mortality in ICU was higher in NSTE-OHCA (54%) than in STE-OHCA (42%) but didn't reach statistical significance. However, at 30 days, mortality was significantly higher in the NSTE-OHCA group than in the STE-OHCA group (57% vs 43% with $p<0,05$).

Our secondary efficacy end points: neurological deficit (Glasgow Coma Scale at the discharge of the ICU), length of stay in the ICU, length of mechanical ventilation, length of inotropic support were similar between groups. Biological criteria showed that STE-OHCA patients had a significant higher median troponin ratio ($p<0,01$), whereas median creatinine level were higher in NSTE-OHCA ($p<0,05$). Our secondary safety end points (severe bleeding, stroke, ventricular tachycardia, and acute kidney failure leading to renal-replacement therapy) were also similar between groups, with no statistical difference (Table 9). NSE levels were unfortunately not comparable due to missing data.

Table 9. Clinical outcomes			
	STE-OHCA (N= 124)	NSTE-OHCA (N=113)	*p-value*
Primary end point			
• Death from any cause in ICU – no. (%)	52 (42)	61 (54)	0,098
• Death from any cause at 30 days – no. (%)	53 (43)	64 (57)	**< 0,05**
Secondary efficacy end points			
• Median length of ICU hospitalization (IQR) – days	4 (2-7)	4 (2-7)	0,80
• Median length of mechanical ventilation (IQR) – days	3 (2-6)	3 (1-6)	0,49
• Median length of inotropic support (IQR) – days	1 (0-1)	0 (0-1)	0,75
• Median score range of Glasgow Coma Scale at discharge in ICU (IQR)	9 (3-15)	8 (3-15)	< 0,55
Biology			
• Median creatinine peak (IQR) - µmol/L	96 (82-140)	120 (94-162)	**<0,05**
• Median troponin peak ratio (IQR)	187 (67-562)	42 (8-111)	**<0,01**
• Median NSE range (IQR) – ng/mL	33 (21-80)*	82 (28-236)*	
Secondary safety end points – no. (%)			
• Severe bleeding	13 (11)	8 (7)	0,48
• Stroke	2 (2)	5 (4)	0,16
• Ventricular tachycardia (VT)	13 (11)	7 (6)	0,25
• Acute kidney failure leading to renal replacement therapy	0 (0)	0 (0)	1,00

* *NSE (more than 80% of missing data)*

UNIVARIATE AND MULTIVARIATE SURVIVAL ANALYSIS

We first performed a univariate analysis in order to focus more on possible items able to predict mortality (Table 10). The univariate analysis showed that the presence of NSTE was associated with a 78% higher mortality risk (OR 1,78 [1,07-2,98], p=0,028).

Some cardiovascular risks increased the mortality risk such as the presence of diabetes mellitus with a 3 to 4 fold increase (OR 3,58 [1,75-7,32], p<0,001), hypertension that doubled the risk (OR 2,12 [1,27-3,56], p=0,005), age and BMI were also associated with a slightly higher risk of mortality (respectively OR 1,03 and 1,06 with p<0,05). Surprisingly, active smoking was not significantly associated with higher risk of mortality (OR 0,75 [0,43-1,28], p=0,29). We observed as well that a previous coronary artery disease was not associated with higher mortality neither (OR 0,97 [0,48-1,99], p=0,94), contrary to a previous peripheral artery disease (OR 4,09 [1,12-14,95], p=0,034). The presence of a shockable first monitored rhythm was significantly decreasing mortality by 83% (OR 0,17 [0,09-0,32], p<0,001), and so was the identification of a culprit lesion at CAG (OR 0,55 [0,33-0,92], p=0,023). Transfer duration to the cath lab or to the ICU did not impact survival in our cohort.

In ICU, the main predictor factors of mortality were the necessity to use inotropic support, the absence of pupillary reflex, and elevated temperature at admission, whereas hypothermia protection measures did not seem to impact patients survival. Acidosis with pH<7,20 and lactic acid elevation were also associated with a higher rate of mortality. Length of ICU hospitalization was associated with a small reduction of mortality (OR 0,93 [0,89-0,98], p=0,005), whereas the length of mechanical ventilation had no significant impact (p=0,93).

We then performed a multivariate analysis by using some of the significant predictable factors from the univariate analysis (age, shockable first monitored rhythm, culprit lesion identified, use of inotropic support). Two factors remained significantly associated with higher mortality: age and the absence of a shockable first monitored rhythm.

Table 10. Estimates of OR and 95% CI of predictors of mortality using linear logistic regression

	OR	95% CI	*p-value*
Univariate analysis			
NSTE-OHCA	1,78	1,07-2,98	**0,028**
Age (years)	1,03	1,018-1,058	**<0,001**
Male sex	0,84	0,47-1,52	0,57
BMI (kg/m^2)	1,06	1,01-1,11	**0,015**
Diabetes mellitus	3,58	1,75-7,32	**<0,001**
Hypertension	2,12	1,27-3,56	**0,005**
Smoking	0,75	0,43-1,28	0,29
Chronic kidney failure	0,68	0,11-4,13	0,68
Previous coronary artery disease	0,97	0,48-1,99	0,94
Previous peripheral artery disease	4,09	1,12-14,95	**0,034**
Shockable first monitored rhythm	0,17	0,09-0,32	**<0,001**
Time from OHCA to coronary angiography (min)	0,99	0,98-1,00	0,30
Time from Cath Lab to ICU (min)	1,00	0,99-1,00	0,60
Culprit lesion identified	0,55	0,33-0,92	**0,023**
Use of inotropic support	1,99	1,19-3,35	**<0,001**
Temperature at admission in ICU (°Celsius)	0,66	0,51-0,84	**0,001**
Hypothermia protection	0,65	0,39-1,09	0,10
Pupillary reflex presence	0,08	0,04-0,18	**<0,001**

Baseline laboratory values at admission in ICU • pH : - pH<7,4 - pH <7,2 • lactic acid – mmol/liter • median troponin ratio • median creatinine – µmol/liter	 2,08 5,98 1.53 1,00 1,02	 0,98-4,39 2,83-12,64 1,30-1,82 0,99-1,00 1,01-1,03	 0,056 **<0,001** **<0,001** 0,08 **<0,001**
Length of ICU hospitalization – days	0,93	0,89-0,98	**0,005**
Length of mechanical ventilation - days	0,99	0,95-1,05	0,93
Multivariate analysis			
Death • NSTE-OHCA • **Age** • **Shockable first monitored rhythm** • Culprit lesion identified • Inotrope support	 1,02 1,03 0,23 0,55 1,61	 0,51-2,02 1,01-1,06 0,11-0,42 0,28-1,09 0,90-2,87	 0,96 **<0,001** **<0,001** 0,09 0,11

NORMAL CAG

We performed a subgroup analysis to study the proportion of normal CAG. Characteristics are reported in Table 12 in the Supplementary data. We observed that normal CAG was mainly observed in younger patients (with a median age at 59 year). There was a lower rate of previous coronary artery disease. The circumstances of cardiac arrest were not different, but there was a trend to a lower number of shockable rhythms in comparison of the rate in STE-OHCA and an equal rate in comparison with NSTE-OHCA group. The post resuscitation ECG showed a STE rate of 30% and a NSTE rate of 70% in this subgroup. No difference in mortality was noted compared to patients with abnormal CAG.

NSTE-CULPRIT LESION-REVASCULARIZATION

In the NSTE-OHCA group, we also studied patients in whom a culprit lesion had been identified and subsequently revascularized. There were only 16 patients (14%) (Figure 2). These patients were older (72 vs 65 year old) and more frequently current smokers (50% vs 29%). The circumstances of cardiac arrest were the same, particularly as to the presence of a shockable rhythm (63% versus 58%). NF and LF durations were similar. Coronary angiography revealed a majority of bitroncular lesions (63%), the majority with circumflex occlusion (50%). Data on ICU admission and hospitalization were comparable. Mortality at 30 days was lower, with a 69% survival rate versus a 43% survival rate in the other NSTE-OHCA patients.

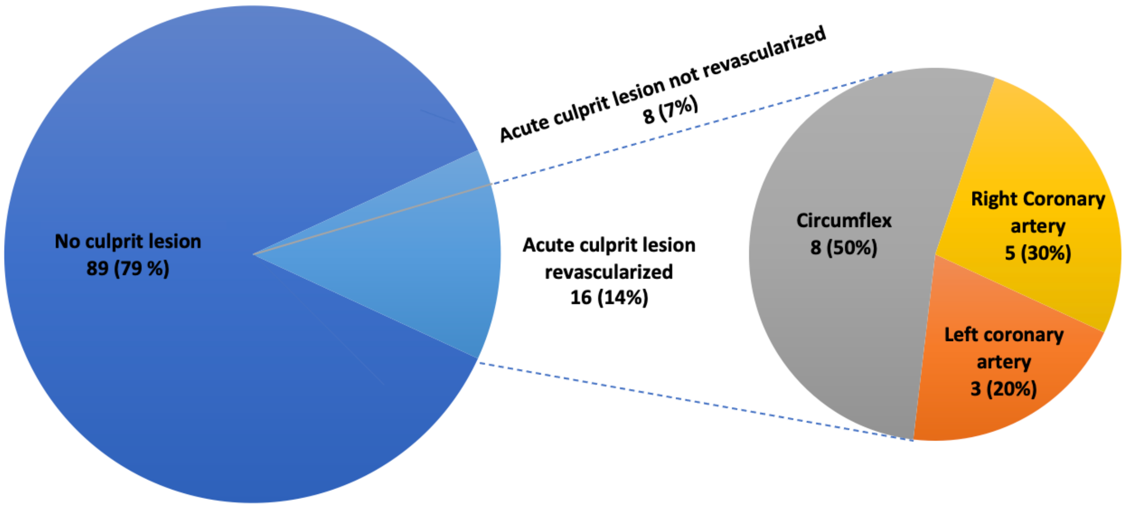

Figure 2. Repartition of culprit lesion in NSTE-OHCA (no. (%))

EVOLUTION BY YEAR

Results were summarized in Supplementary Data (Figure 3-4). Results by year during the last decade showed that number of patients who underwent an emergency CAG after resuscitated OHCA is decreasing, with a majority of STE on ECG, which is consistent with recent recommendations.

Discussion

The aim of our study was to review our practice at University of Tours in the last 10 years regarding the management of OHCA with suspected cardiac cause and to define the place and timing of coronarography in these situations.

237 patients were included during the last decade, and our management of OHCA evolved along the years with the new recommendations based on randomized studies. Our cardiology center is the only center with a cath lab and extracorporeal circulation system of a 600 000 inhabitants area. Thus, it plays a major role in the management of OHCA. Our results are consistent with the published data, as our cohort includes young patients, mostly men, with high cardiovascular risk and are similar to the characteristics of randomized trials[20–22] (COACT, TOMAHAWK, EMERGE). Our mortality rate at 30 days is 49% in the cohort, 43% in STE-OHCA group and 57% in NSTE-OHCA group. These results are in keeping with the 50% mortality rate at 30 days observed in the TOMAHAWK[21] study, the 35% mortality rate observed at 90 days in the COACT study, and the 65% observed at 180 days in the EMERGE[22] study.

The analysis of our results by year shows a shift in our practice: less coronarography over the years for the same incidence of OHCA (with an exceptional decrease in 2020 and 2021, years of COVID worldwide pandemic). That may be explained by the release of new recommendations based on randomized trials favoring emergency CAG in patients with shockable heart rhythm and/or STE on ECG. Indeed, the latest studies and lately, the *Heyne and al* meta-analysis[23], did not manage to prove a benefit of an immediate versus a delayed CAG in OHCA without ST segment elevation leading to new recommendations.

Our results are consistent with the release of these new recommendations as we observed since 2019 a clear decrease of immediate CAG in patients with NSTE-OHCA and an increase of immediate CAG in STE-OHCA, with almost exclusively immediate CAG in STE-OHCA patients since 2019. Actual recommendations are based on 3 parameters: medical history, shockable rhythm and post resuscitation ECG. Data regarding previous medical history and cardiac symptoms before cardiac arrest are often difficult to collect in the setting of an emergency intervention for OHCA in unconscious patients with witnesses that may have no knowledge of the patients when there is a witness. Our 10-year retrospective study was unable to reliably retrieve these data.

Our analysis of post resuscitation ECG revealed the following information: ECG post resuscitation showed mostly narrow QRS, with localized ischemia in a coronary territory, mostly anterior and inferior in STE-OHCA, and lateral territory in NSTE-OHCA. In the NSTE-OHCA group, the ECG study showed a significantly higher rate of bundle-branch block. "Ischemic" signs are more diffuse and more lateral, which may be explained by the presence of a bundle branch block (BBB) and its secondary repolarization trouble. Some studies show that the appearance of a BBB on post resuscitation ECG could be linked to non-specific secondary repolarization disorders due to CPR management (metabolic disorders post cardiac arrest, use of adrenaline…) which render the interpretation of these NSTE "ischemia sign" complicated[26–30].

Coronary angiography revealed a culprit lesion in 72% of STE-OHCA, with revascularization of this lesion in 66% of cases, almost exclusively by angioplasty at the same time as the diagnostic coronary angiography, whereas only 21% of NSTE-OHCA had a culprit lesion (and only 14% were revascularized, the others were treated medically). Early revascularization has been proven beneficial in reducing mortality, particularly in the most severe situations, such as cardiogenic shock[24,25]. It probably explains the significant difference in mortality between the STE-OHCA and NSTE-OHCA groups in patients with similar severity at the admission to the ICU.

Moreover, the coronary angiography lesions showed a coronary lesion rate equivalent in terms of bitroncular or tritroncular lesions in both groups (around 25% in STE-OHCA and 20% in NSTE-OHCA), but with more frequently chronic lesions incidentally discovered, since the rate of acute culprit lesions was only 21%. The chronic lesions rate in our study needs to be analyzed with the characteristics of the NSTE group in mind. These patients were older with a higher cardiovascular risk (significantly more comorbidities, especially more kidney failure with GFR < 30 mL/min). These patients had more frequently a previous history of coronary artery disease, which probably influenced the decision to undergo immediately coronary angiography, as they have a significantly higher rate of sudden death than in general population. In our retrospective study, we found a 21% rate of culprit lesion in NSTE-OHCA group, which was similar to the culprit lesion rate in TOMAHAWK (23%), in PROCRAT II[12] (29%) and in Spirito and al[31] (20%).

Our multivariate analysis showed that older age and absence of shockable first monitored rhythm were significantly associated with mortality rendering NSTE-OHCA no longer significantly associated with mortality. Our results are consistent with the PROCRAT II study that showed that one of the predictive factors of favorable outcome in OHCA without STE was an initial shockable rhythm ($p<0.001$)[12] and that it was the sole independent indicator for PCI requirement in that population. The conclusion of PROCRAT II study was that CAG and PCI strategy in OHCA without STE was beneficial mostly for men older than 50 years old, with a shockable first rhythm monitored. Spirito *and al* found that chest pain or shockable first rhythm monitored were also two predictable factors associated in OHCA without STE to patients with acute coronary lesion. These observations strengthen the hypothesis that a first shockable monitored rhythm in NSTE-OHCA may help predict the presence of a culprit acute coronary lesion in the patients that is likely to benefit from an emergency CAG and subsequent PCI.

Our data confirm these findings. There was a higher and significant rate of shockable rhythm in the STE-OHCA group (77%), and a similar rate of shockable rhythm in the NSTE-OHCA group (58% vs 57% in the TOMAHAWK study). However, other studies found a weak correlation between the presence of a shockable rhythm and coronary occlusion (only 23%)[32]. In the Yannopaulos study, the culprit lesion rate rose to 64% only if there were more than 3 External Electric Shock (EECs)[33]. Besides, regarding the association between shockable first rhythm monitored and mortality in OHCA without STE, the COACT study who only included OHCA with a shockable rhythm did not prove any benefice on survival of an immediate versus delayed CAG in that population. We conclude that the presence of a shockable first rhythm monitored remains a debatable factor for the indication of emergency CAG in the NSTE-OHCA population.

To further assess the question of OHCA patients' prognosis, especially in NSTE-OHCA where survival was lower at 30 days, we studied the transfer delays between the initial OHCA location and the cath lab and from the cat lab to the ICU in our center. This because in cardiac arrest, neurological outcome is known to be linked to the early initiation of neuroprotection management. Indeed, neurological deficit remains a major preoccupation in intensive care management of OHCA patients, and this, whatever the final cause of cardiac arrest. Sooner is started neuroprotection management, better is the outcome with a lower neurological deficit[34]. Neuroprotection management is possible at every stage of the CPR process, from the very beginning of CPR to the hospitalization in ICU (maintaining cerebral circulation, regulation of the PO_2/PCO_2 and temperature in ICU…). During the last decade, hypothermia played a major role in neuroprotection at the beginning. However, its benefit versus normothermia[35,36] had been questioned and was progressively less used in ICU. In our study, even if low temperature at admission seemed to be associated with a better outcome (OR 0,66 [0,51-0,84], p=0,001) during the ICU hospitalization, hypothermia protection was not associated with a better survival (OR 0,65 [0,39-1,09], p=0,10). We did observe that trend in our study with a lower rate of hypothermia measures since 2015. Moreover, in our center, the cath lab and the medical ICU are in different areas of the city. We observed a median transfer time of 100 min in our cohort that results both from the distance and the needs to mobilize considerable health care resources to organize the medical transfer of ventilated patients that are sometimes unstable. This may have delayed the initiation of neuroprotection maneuvers. This delayed neuroprotection management may partly explain the increase of mortality in NSTE-OHCA group, even if in our study, time of transfer from cath lab to ICU was not a predictor factor of death (OR 1,00 [0,99-1,00], p=0,60).

Concerning our secondary end point criteria (Glasgow Coma Scale at the discharge of the ICU), length of stay in the ICU, length of mechanical ventilation, length of inotropic support and our secondary safety end points severe bleeding, stroke, ventricular tachycardia, and acute kidney failure leading to renal-replacement therapy, there were no statistical difference between STE-OHCA and NSTE-OHCA. If we compare the incidence of these events in our NSTE-OHCA cohort with the COACT and TOMAHAWK results, we had similar proportions of VT events (<10%), severe bleeding (5%) and stroke (4-5%). We did observe a much lower incidence of acute kidney failure leading to renal-replacement therapy (<1% versus 15% in TOMAHAWK), that maybe partly explained by short CAG procedures with low amounts of contrast dye injection. This relatively low severe bleeding rate is probably due to the predominance of radial access in our center over the last 20 years. The radial punction is preferred for STE group without OHCA in general, as it has been showed that radial punction reduced mortality [37]. One of our major discordances with these two studies is the mean GSC Scale at discharge of ICU. Published studies report a mean of 15, whereas we observed a median GSC in both groups around 8-9 which could be explained by the inclusion of patients pronounced deceased at the discharge of ICU.

Finally, our data in NSTE-OHCA group seem to support the findings of randomized studies (COACT, TOMAHAWK, EMERGE, DISCO) that immediate coronary angiography did not prove a benefit in overall mortality. We stepped our study further by analyzing data from patients with normal coronary angiography. Indeed, if the cause of cardiac arrest is well identified as a non-ischemic cause, the futility of coronary angiography seems obvious. However, as previously discussed, the clinical history is often difficult to collect, as well as a normal ECG does not predict a 100% rate of normal CAG[38]. Recent scores tried to predict acute coronary lesion post OHCA (for example, ASC2 score based on the presence of angina, congestive heart failure, a shockable arrest rhythm or the presence of ST elevation in two contiguous leads), but the results were unfortunately no reliable without ST segment elevation[39]. Patients with a normal CAG were younger patients, aged 59, with an equivalent sex ratio and cardiovascular risk. Regarding the circumstances of cardiac arrest, there was no significant difference except for shockable rhythm. We observed less shockable rhythms in patients with normal CAG than in patient with STE, but equal proportion of shockable rhythm in comparison with NSTE . Post-resuscitation ECG showed a STE rate of 30%, resulting in a false positive in one third of cases, confirming the limited reliability of ECG alone.

The next question to assess in out-of-hospital cardiac arrest is to identify more precisely criteria in NSTE-OHCA patients, helping us to select with a high specificity and sensibility patients with acute coronary syndrome and culprit lesion, for whom coronary angiography and early revascularization may then be beneficial. The data from our study should be treated with caution due to the small number of patients involved, but we have noted an improvement in survival of almost 70%.

However, our study has some limits that need to be acknowledged. It is a retrospective study that involves missing data, especially on neurologic evaluation in ICU (NSE, pupillary reflex, time to hypothermia…). We had to use the Glasgow Coma Scale at the discharge of ICU, despite its imprecision, and the fact that it was not validated for neurological evaluation after OHCA. CPC scale used in most studies[20,21] was not retrospectively available for our cohort. Post resuscitation ECG could not be all re-analyzed as some of them were not scanned in patient's file. The investigators relied on ECG analysis reported in patients' medical files. It could result in some misinterpretation in ECG analysis, even if most ECG analysis retrieved in the electronic files were performed by experienced cardiologists.

In conclusion, there is still a long way to go to precisely define the subgroup of patients in which immediate CAG after OHCA without STE, would be benefic and with a good neurological predictable outcome, in order to be harmless and avoid futile interventions[40,41].

By using a combined score, *Pareek and al*[42] showed that immediate CAG is associated with improved neurological outcome in selected patients with OHCA with favorable prediction of neurologic recovery (MIRA- CLE2 score 0-3), cardiogenic shock (SCAI grades B-E), or STEMI on arrival. Unfortunately, immediate CAG, good neurological outcome and NSTEMI were not significantly associated. Futures trials are coming in order to further answer the question of the optimal treatment and timing of CAG in NSTE-OHCA[43].

Conclusion

Management of OHCA patients with cardiac cause (ST elevation or No ST elevation) in our center during the last decade is consistent with the recent recommendations. Our results confirm the need for coronary angiography in patients with STE and the lack of benefit of emergency coronary angiography in patients without STE on post ECG resuscitation. Future work should focus on identifying no-STE patients with acute coronary lesions that may benefit from emergency CAG and subsequent revascularization.

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Supplementary data

Table 11. Comparison of daily medical treatment of the patients			
	STE-OHCA (N= 124)	NSTE-OHCA (N=113)	*p-value*
Aspirin – no. (%)	17 (14)	27 (24)	**<0,05**
P2Y12 – no. (%)	7 (6)	14 (12)	0,110
Vitamin K antagonist (VKA) – no. (%)	6 (5)	14 (12)	0,063
Direct oral anticoagulant (DOAC) – no. (%)	3 (2)	6 (5)	0,316
Renin-angiotensin system (RAS) inhibitors – no. (%)	29 (23)	48 (42)	**<0,01**
Beta-blockers – no. (%)	27 (22)	38 (34)	**<0,05**
Metformin – no. (%)	9 (7)	13 (12)	0,367

Table 12. Summary of all the different groups and subgroups					
	COHORT (N= 237)	STE-OHCA (N= 124)	NSTE-OHCA (N=113)	NORMAL CAG (N= 74)	NSTE-OHCA with revascularized culprit lesion (N=16)
Characteristics of the patients					
Median age (IQR) – year	63 (51-71)	58 (50-69)	65 (55-76)	59 (45-69)	72 (62-81)
Male sex – no. (%)	180 (76)	94 (76)	83 (73)	49 (66)	14 (88)
Median BMI (IQR) – kg/m^2	27 (24-31)	27 (24-31)	27 (23-30)	25 (22-31)	28 (25-30)
Cardiovascular risk – no. (%)					
• Diabetes mellitus	45 (19)	18 (15)	26 (2)	16 (22)	2 (13)
• Hypertension	114 (48)	55 (45)	58 (51)	29 (39)	9 (56)
• Current smoker	79 (33)	45 (37)	33 (29)	22 (30)	8 (50)
• Dyslipidemia	76 (32)	33 (27)	42 (37)	18 (24)	7 (44)
Previous coronary artery disease – no. (%)	35 (15)	12 (10)	23 (20)	2 (3)	4 (25)
Previous peripheral artery disease – no. (%)	14 (6)	5 (4)	9 (8)	4 (5)	1 (6)
Previous PCI – no. (%)	26 (11)	8 (7)	18 (16)	2 (3)	4 (25)
Previous CABG – no. (%)	7 (3)	2 (2)	5 (4)	0 (0)	0 (0)
Previous stroke – no. (%)	9 (4)	7 (6)	2 (2)	0 (0)	1 (6)

Previous heart surgery – no. (%)	8 (3)	4 (3)	4 (4)	3 (4)	1 (6)
Chronic kidney failure – no. (%)	5 (2)	0 (0)	5 (4)	1 (1)	1 (6)
Chronic respiratory failure – no. (%)	1 (0,4)	0 (0)	1 (1)	1 (1)	0 (0)
Daily medical treatment of the patients					
Aspirin – no. (%)	45 (19)	17 (14)	27 (24)	11 (15)	2 (13)
P2Y12 – no. (%)	21 (9)	7 (6)	14 (12)	2 (3)	3 (19)
Vitamin K antagonist (VKA) – no. (%)	20 (8)	6 (5)	14 (12)	9 (12)	2 (13)
Direct oral anticoagulant (DOAC) – no. (%)	9 (4)	3 (2)	6 (5)	3 (4)	2 (13)
Renin-angiotensin system (RAS) inhibitors – no. (%)	78 (33)	29 (23)	48 (42)	21 (28)	8 (50)
Beta-blockers – no. (%)	66 (28)	27 (22)	38 (34)	22 (30)	3 (19)
Metformin – no. (%)	22 (9)	9 (7)	13 (12)	8 (11)	1 (6)
Cardiac arrest circumstances					
Arrest witnessed – no. (%)	200 (84)	104 (84)	92 (81)	59 (80)	14 (88)
Bystander CPR – no. (%)	174 (73)	95 (77)	75 (66)	51 (69)	13 (81)
Shockable first monitored rhythm – no. (%)	163 (69)	95 (77)	65 (58)	43 (58)	10 (63)
Median time from	5 (1-10)	3 (0-5)	5 (1-10)	5 (1-10)	1 (0-7)

• arrest to basic life support **(No Flow)** (IQR) – min • arrest to return of spontaneous circulation **(Low Flow)** (IQR) – min	20 (13-27)	20 (12-28)	20 (14-30)	20 (12-25)	20 (15-34)
Procedures, treatments, and characteristics of coronary artery disease					
Median time from arrest to coronary angiography (IQR) – min	135 (107-170)	135 (110-174)	130 (100-161)	130 (100-153)	121 (91-152)
Median time of coronary angiography (IQR) – min	19 (11-33)	25 (14-39)	15 (10-20)	12 (9-18)	31 (46)
Catheterization access – no. (%) • Radial • Femoral	192 (81) 41 (17)	98 (79) 23 (19)	90 (80) 18 (16)	60 (81) 13 (18)	15 (94) 1 (6)
Severity of coronary artery disease – no. (%) • No significant disease • One-vessel disease • Two-vessel disease • Three-vessel disease	75 (32) 159 (67) 60 (25) 54 (23)	23 (19) 39 (32) 32 (26) 29 (23)	50 (44) 22 (20) 22 (20) 15 (13)	74 (100) / / /	0 (0) 4 (25) 10 (63) 2 (13)
Culprit lesion identified – no. (%)	115 (49)	89 (72)	24 (21)	/	16 (100)
Revascularization treatment – no. (%) • PCI performed • CABG • Pharmacologic or conservative treatment	101 (43) 1 (0,4) 59 (25)	82 (66) 1 (1) 18 (15)	16 (14) 0 (0) 39 (35)	/ / /	16 (100) 0 (0) 0 (0)
Median amount of contrast dye (IQR) – mL	145 (52-120)	92 (61-132)	63 (50-99)	52 (41-66)	127 (112-164)

Baseline characteristics at admission in ICU					
Median time from cath lab to ICU transfer (IQR) - min	100 (77-123)	100 (82-127)	90 (70-120)	96 (75-120)	90 (67-115)
Median score range of Glasgow Coma Scale at admission in ICU (IQR)	3 (3-3)	3 (3-3)	3 (3-3)	3 (3-3)	3 (3-3)
Median blood pressure (IQR) – mmHg • Systolic • Diastolic • PAM	 121 (101-140) 71 (60-85) 87 (75-101)	 121 (103-136) 72 (64-85) 89 (78-100)	 121 (103-140) 68 (60-84) 87 (75-103)	 118 (99-130) 67 (59-80) 83 (71-97)	 132 (117-149) 67 (60-77) 86 (83-102)
Inotropic support – no. (%)	120 (51)	66 (53)	52 (46)	38 (50)	6 (38)
Median temperature (IQR) – °Celsius	35,4 (34,5-36,1)	35,5 (34,6-36,2)	35,3 (34,4-36)	35,6 (34,5-36,2)	35,2 (34,7-35,8)
Median left ventricular ejection fraction (IQR) - %	40 (30-50)	40 (30-50)	45 (30-60)	45 (30-60)	45 (40-50)
Baseline laboratory values • pH • Median lactic acid (IQR) – mmol/liter • Median partial pressure of O_2 (IQR) – kPa • Median partial pressure of CO_2 (IQR) – kPa • Median creatinine (IQR) – µmol/liter • Median troponin ratio (IQR) • Median hemoglobin (IQR) – (g/dl)	 7,31 (7,23-7,37) 2,5 (1,5-4,2) 118 (83-193) 41 (38-46) 98 (81-124) 43 (10-119) 13,7 (12,4-14,9)	 7,32 (7,25-7,37) 2,4 (1,4-3,9) 111 (82-165) 41 (37-47) 94 (78-114) 79 (25-181) 13,6 (12,5-15,1)	 7,29 (7,20-7,36) 2,7 (1,8-4,3) 140 (93-227) 43 (38-46) 105 (90-134) 14 (5-53) 13,8 (12,2-14,8)	 7,29 (7,21-7,37) 2,7 (1,5-4,9) 120 (87-196) 41 (37-46) 103 (82-135) 10 (4-47) 13,5 (12,2-14,7)	 7,33 (7,21-7,36) 2 (1,8-2,4) 42 (40-46) 23 (21-36) 109 (96-128) 19 (10-78) 13,6 (12,8-14,3)
Hypothermia protection – no. (%)	118 (50)	58 (47)	59 (52)	35 (47)	8 (50)
Pupillary reflex presence – no. (%)	138 (58)	71 (57)	66 (58)	38 (51)	13 (81)

Clinical outcomes					
Primary end point					
• Death from any cause in ICU – no. (%)	112 (47)	52 (42)	61 (54)	41 (55)	5 (31)
• Death from any cause at 30 days – no. (%)	117 (49)	53 (43)	64 (57)	43 (58)	5 (31)
Secondary efficacy end points					
• Median length of ICU hospitalization (IQR) – days	4 (2-7)	4 (2-7)	4 (2-7)	4 (2-7)	7 (2-15)
• Median length of mechanical ventilation (IQR) – days	3 (1-6)	3 (2-6)	3 (1-6)	3 (2-5)	5 (2-8)
• Median length of inotropic support (IQR) – days	1 (0-1)	1 (0-1)	0 (0-1)	1 (0-1)	0 (0-1)
• Median score range of Glasgow Coma Scale at discharge in ICU (IQR)	6 (3-15)	9 (3-15)	8 (3-15)	3 (3-15)	14 (3-14)
Biology					
• Median creatinine peak (IQR) - µmol/L	104 (83-150)	96 (82-140)	120 (94-162)	102 (82-135)	113 (101-192)
• Median troponin peak ratio (IQR)	89 (22-297)	187 (67-562)	42 (8-111)	19 (3-71)	19 (3-71)
• Median NSE range (IQR) – ng/mL	40 (23-161)*	33 (21-80)*	82 (28-236)*	40 (23-161)*	27 (22-28)*
Secondary safety end points – no. (%)					
• Severe bleeding	21 (9)	13 (11)	8 (7)	8 (11)	2 (12)
• Stroke	8 (3)	2 (2)	5 (4)	3 (4)	2 (12)
• Ventricular tachycardia	21 (9)	13 (11)	7 (6)	3 (4)	2 (12)
• Acute kidney failure leading to renal replacement therapy	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)

**NSE (more than> 80% of missing data)*

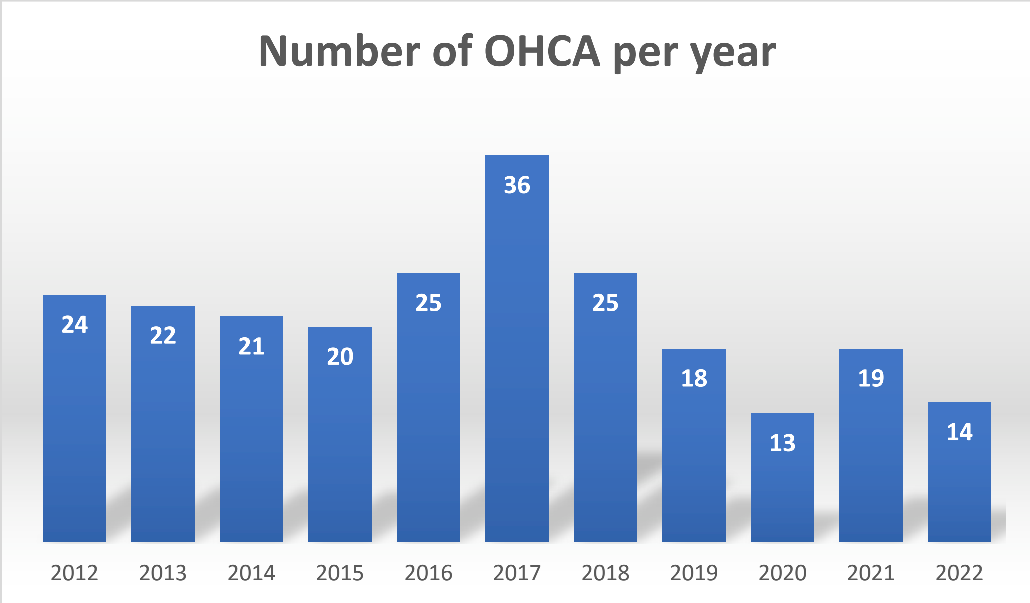


Figure 3. Number of OHCA per year in our center from 2012 to 2022

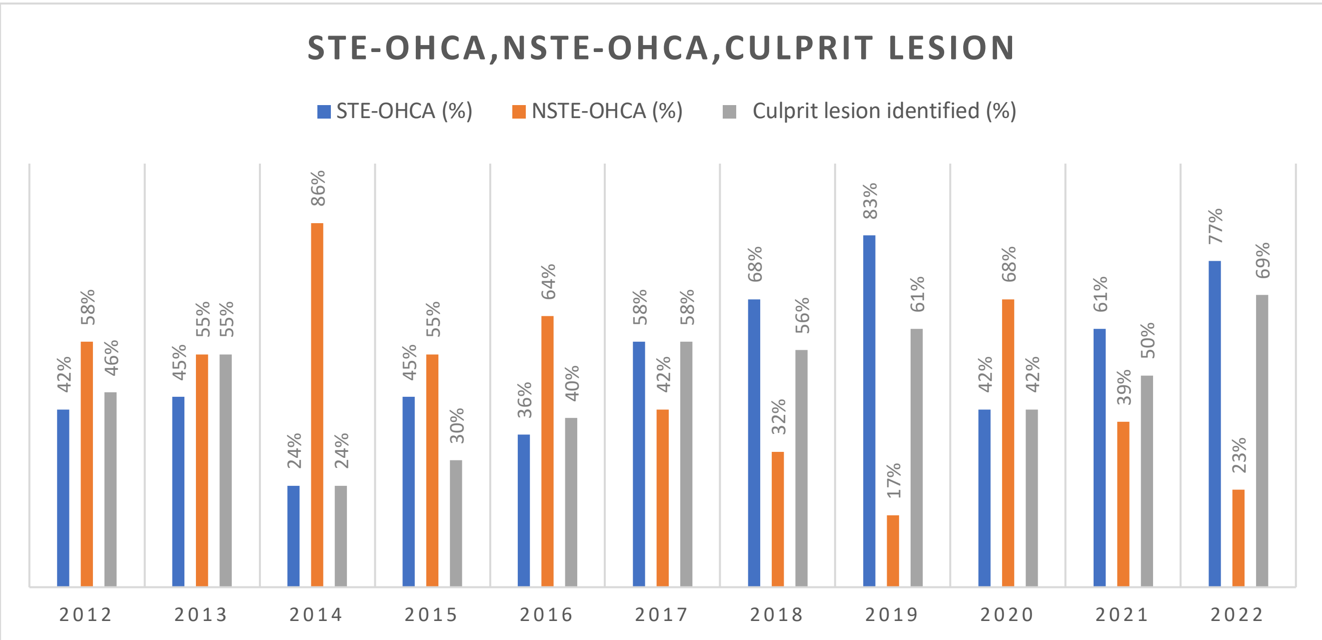
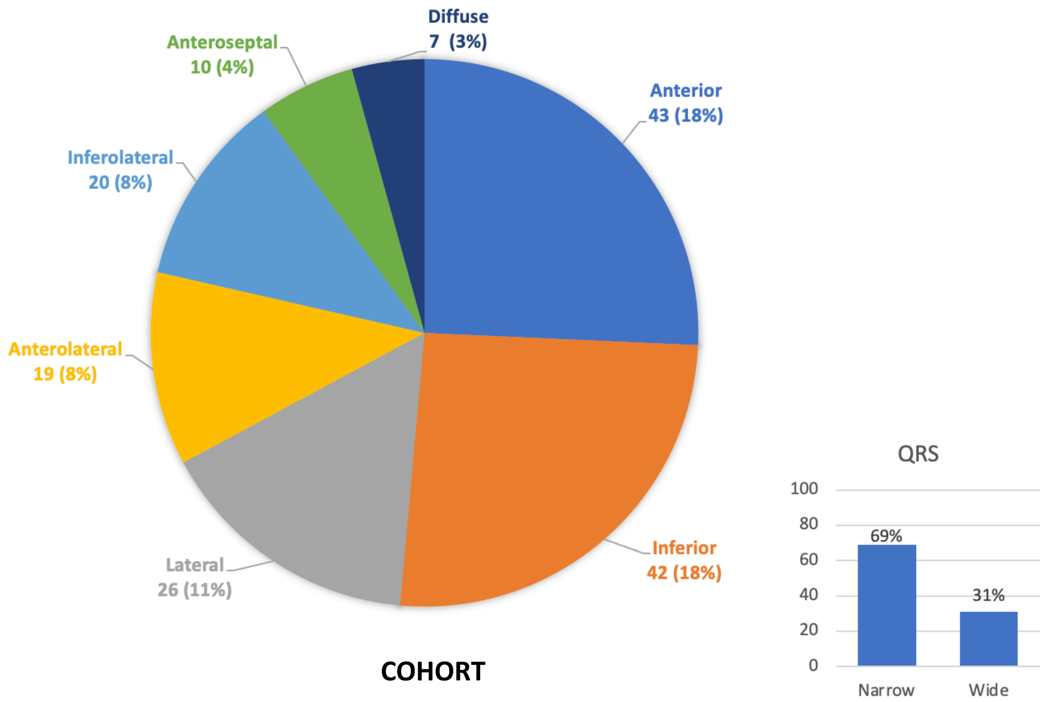
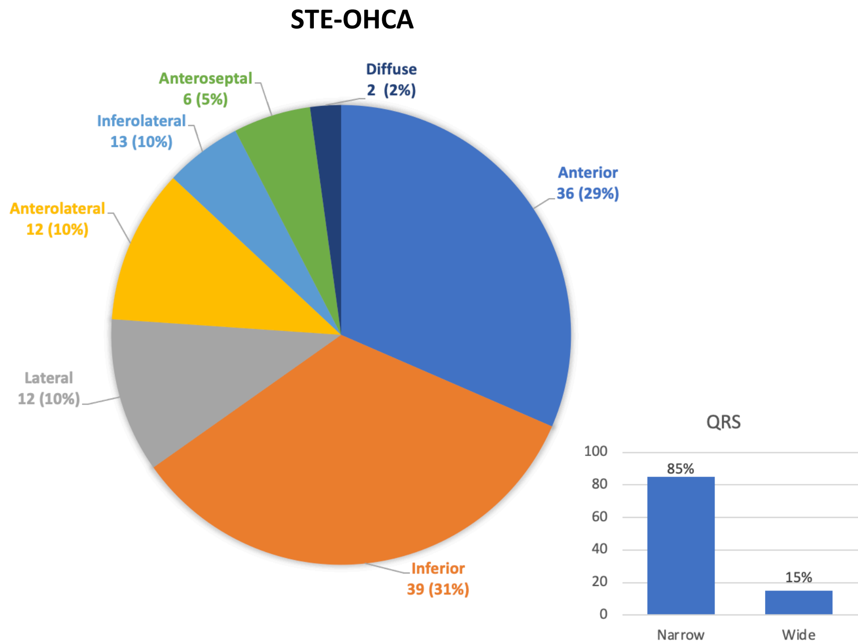


Figure 4. Evolution during the years of percentage of STE-OHCA, NSTE-OHCA and culprit lesion





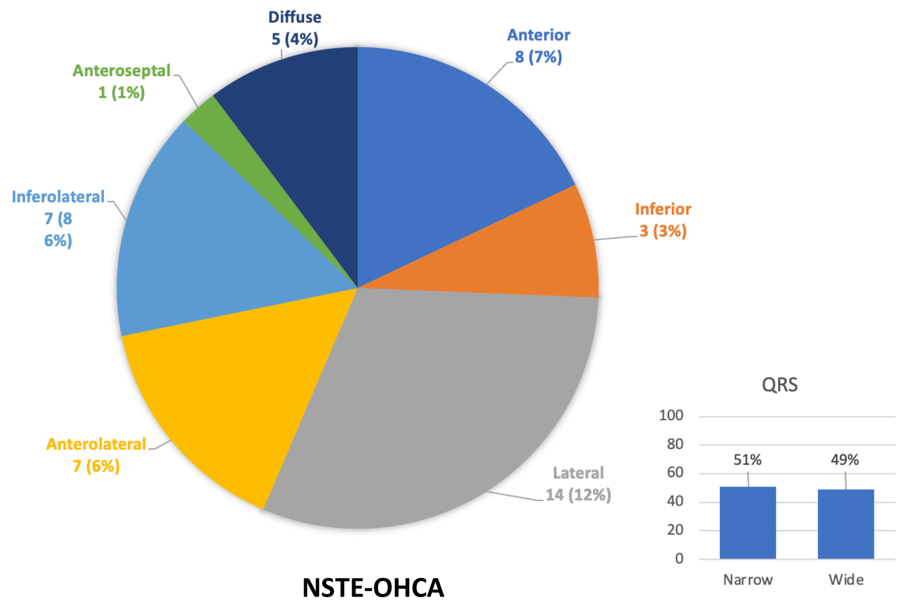
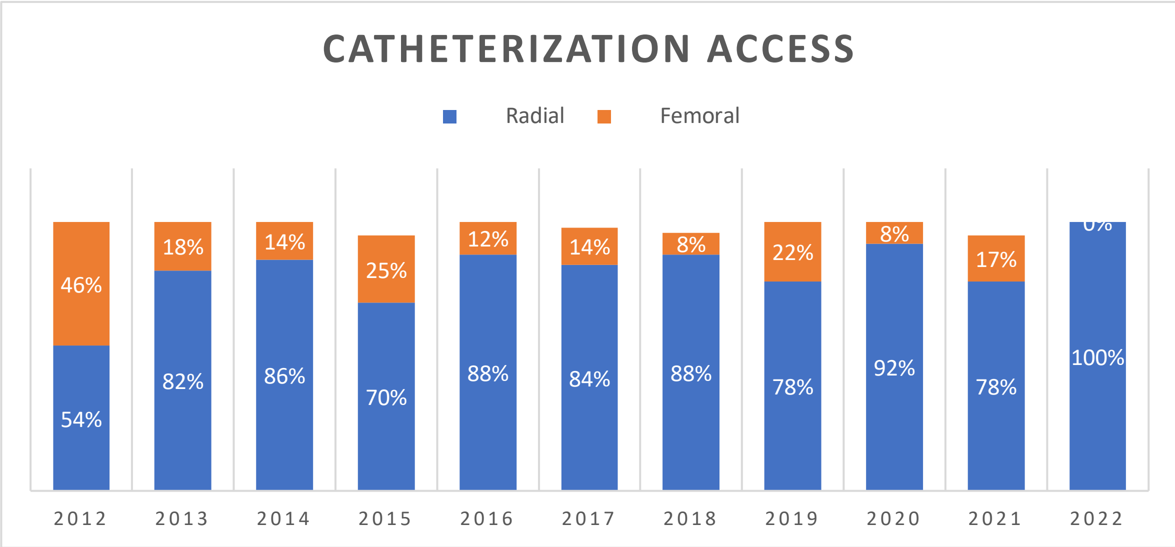
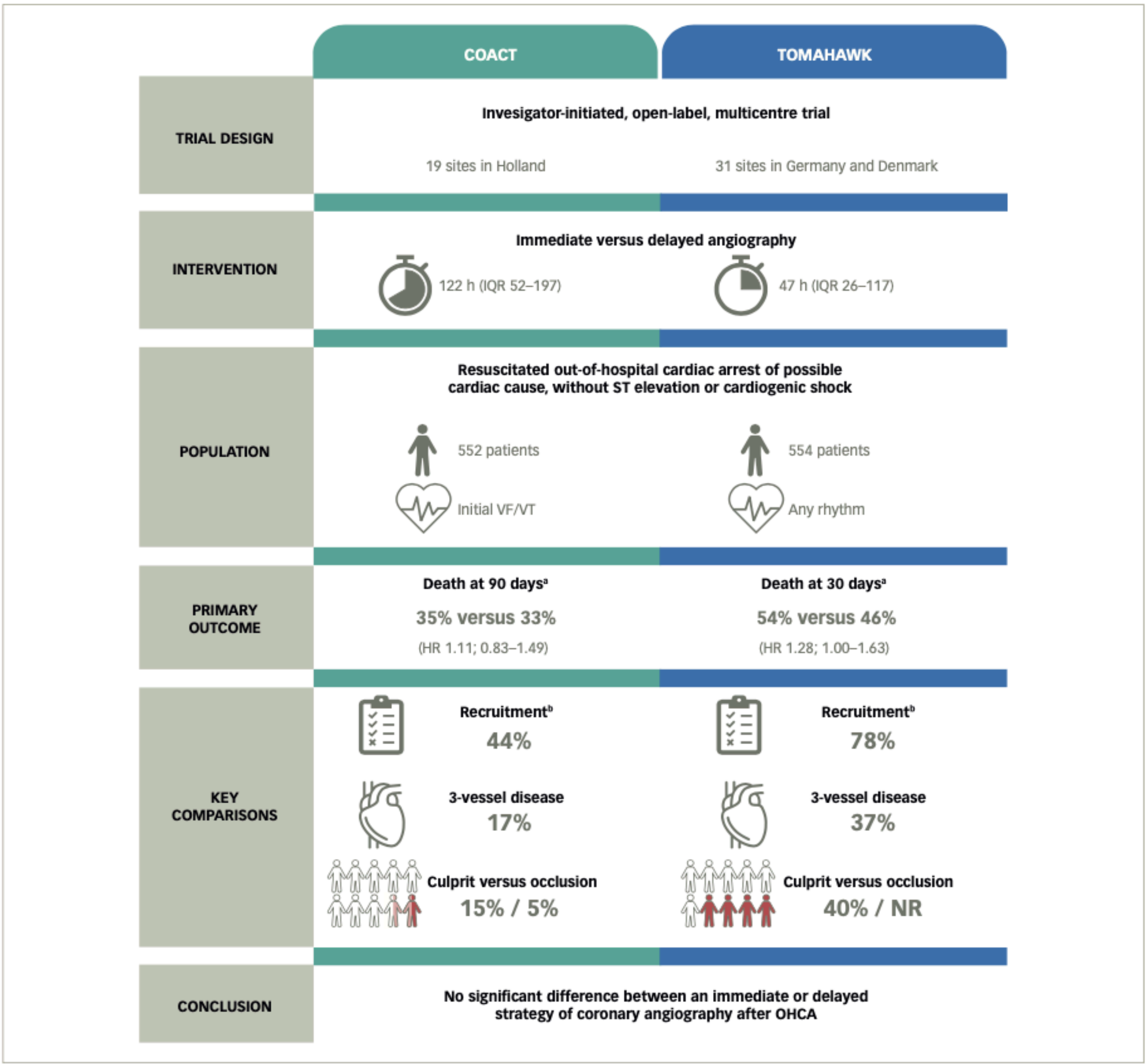


Figure 5. Localization of ischemia on ECG in our cohort and STE and NSTE-OHCA subgroups from 2012 to 2022

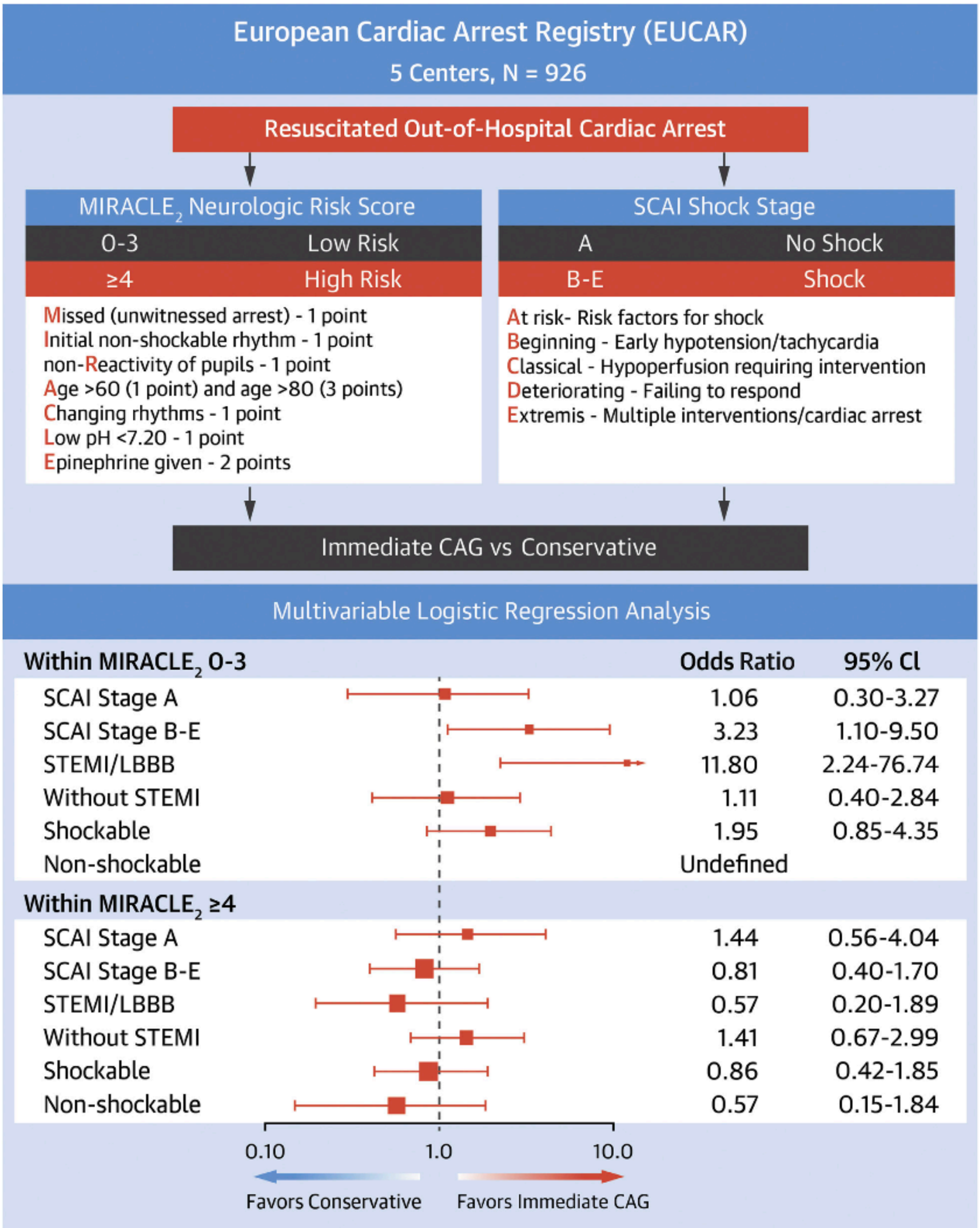


<u>Figure 6. Evolution of catheterization access from 2012 to 2022</u>



Time to delayed angiography specified as median.
[a]The primary outcome in COACT was survival at 90 days, the hazard ratio for death is provided to allow comparison between trials. [b]Complete screening logs were not maintained for either trial. The recruitment rate was calculated from the screening samples and based on the number of patients that were eligible for the trial after all inclusion (including informed consent for COACT) and exclusion criteria were applied.
h = hours; HR = hazard ratio; IQR = interquartile range; NR = not recorded; OHCA = out-of-hospital cardiac arrest; VF = ventricular fibrillation; VT = ventricular tachycardia.

Figure 7. Comparison of COACT and TOMAHWAK trials issued from Wa and al[43]

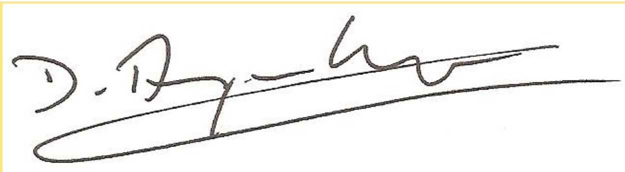


Pareek N, et al. *J Am Coll Cardiol Intv.* 2022;15(10):1074-1084.

CAG = coronary angiography; LBBB = left bundle branch block; SCAI = Society for Cardiovascular Angiography and Interventions; STEMI = ST-segment elevation myocardial infarction.

Figure 8. Results of the MIRACLE$_2$ Score and SCAI Grade to Identify Patients With Out-of-Hospital Cardiac Arrest for Immediate Coronary Angiography[42]

Vu, le Directeur de Thèse Pr Denis Angoulvant



Vu, le Doyen

De la Faculté de Médecine de Tours,

LANDWERLIN Charlène
58 pages – 12 tableaux – 8 figures

Résumé :
L'infarctus du myocarde est une des principales causes d'arrêt cardiaque extrahospitalier (ACR-EH). Cependant, la place de la coronarographie en urgence reste incertaine dans le cas des ACR-EH sans sus-décalage du segment ST. Ni les dernières recommandations de l'ESC ou de l'ACC/AHA, ni les essais randomisés (COACT, TOMAHAWK, EMERGE) n'ont réussi à définir clairement le délai et le bénéfice de la coronarographie en urgence sur la mortalité dans ce sous-groupe d'ACR-EH.
Notre étude rétrospective réalisée au CHU de Tours, de janvier 2012 à décembre 2022, étudie l'impact clinique de la réalisation immédiate de la coronarographie chez les patients ayant fait un ACR-EH avec ou sans ST+ à l'électrocardiogramme.
Notre étude a inclus tous les patients pris en charge pour ACR-EH (237 patients) et adressés directement en salle de coronarographie après récupération d'une activité cardiaque, répartis en deux sous-groupes : ACR-EH-ST+ (124 patients) et ACR-EH-ST- (113 patients). A 30 jours, 43% des patients du groupe ACR-EH-ST+ et 57% des patients du groupe ACR-EH-ST- étaient décédés ($p<0,05$). La réalisation en urgence de la coronarographie post ACR-EH retarde l'admission en réanimation et la mise en place des mesures de neuroprotection (temps moyen de transfert de 100 minutes), ce qui pourrait expliquer en partie la surmortalité du groupe ACR-EH-ST-, chez qui seulement 21% de lésions coronaires coupables ont été retrouvées. L'âge et l'absence d'un rythme choquable sont les seuls facteurs qui restent statistiquement associés à la mortalité en analyse multivariée dans notre étude.
La prise en charge des ACR-EH suspects d'une étiologie cardiaque dans notre centre est cohérente avec les résultats des dernières recommandations et études. Le recours à la coronarographie en urgence semble plus bénéfique chez les patients avec sus décalage post ressuscitation. Notre étude n'a pas réussi à identifier de nouveaux facteurs prédictifs permettant de mieux sélectionner les patients avec un ST- post ACR-EH pouvant bénéficier de la coronarographie en urgence avant l'admission en réanimation.

Mots clés : Arrêt cardiaque extrahospitalier, sous-décalage, sus-décalage, coronarographie, lésion coronaire coupable, neuroprotection, mortalité toute cause

Jury :

Président du Jury :	Professeur Fabrice IVANES
Directeur de thèse :	Professeur Denis ANGOULVANT
Membres du Jury :	Professeur Antoine GUILLON
	Docteur Christophe SAINT ETIENNE
	Docteur Maëva GOURRAUD

Date de soutenance : 7 septembre 2023