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Clinical paper

Causes of in-hospital cardiac arrest – Incidences and rate of recognition☆

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ABSTRACT

Background and methods: Do emergency teams (ETs) consider the underlying causes of in-hospital cardiac arrest (IHCA) during advanced life support (ALS)? In a 4.5-year prospective observational study, an aetiology study group examined 302 episodes of IHCA. The purpose was to investigate the causes and cause-related survival and to evaluate whether these causes were recognised by the ETs.
Results: In 258 (85%) episodes, the cause of IHCA was reliably determined. The cause was correctly recognised by the ET in 198 of 302 episodes (66%). In the majority of episodes, cardiac causes (156, 60%) or hypoxic causes (51, 20%) were present. The cause-related survival was 30% for cardiac aetiology and 37% for hypoxic aetiology.

The initial cardiac rhythm was pulseless electrical activity (PEA) in 144 episodes (48%) followed by asystole in 70 episodes (23%) and combined ventricular fibrillation/ventricular tachycardia (VF/VT) in 83 episodes (27%). Seventy-one patients (25%) survived to hospital discharge. The median delay to cardiopulmonary resuscitation (CPR) was 1 min (inter-quartile range 0–1 min).
Conclusions: Various cardiac and hypoxic aetiologies dominated. In two-thirds of IHCA episodes, the underlying cause was correctly identified by the ET, i.e. according to the findings of the aetiology study group.

1. Introduction

If cardiac arrest (CA) occurs in a hospitalised patient, the primary intervention is cardiopulmonary resuscitation (CPR) following the current advanced life support (ALS) guidelines, which include a reminder of the causes of CA through the mnemonic "4H4T" (hypoxia, hypovolaemia, hypo-/hyperkalaemia, hypothermia, thrombosis/pulmonary emboli, tamponade cardiac, toxins, tension pneumothorax).[1,2] Exactly how often an IHCA episode with one of the 4H4T causes occurs is unknown. One may debate how aggressive the causes of arrest should be sought by the alerted emergency team (ET) during CPR, as this may interfere with the quality of resuscitation efforts. Few studies have investigated to what degree the ETs actually recognise the causes of arrest during ALS.

Despite systematic research on the aetiology and its influence on outcomes after CA and the recommendations to prevent CA by recognising clinical deterioration in sick patients, the incidence of IHCA has remained largely unchanged.[3–9] However, an increase in survival has been demonstrated in hospitals working with strategic improvements in the "chain-of-survival" (COS).[10,11] For the post-arrest care of initial survivors, knowledge and treatment of the underlying chronic and acute medical conditions may be important.[12] We believe a prospective observational study is needed to elucidate whether the causes of arrest are investigated during ALS.

This study was conducted to investigate the cause-specific incidences of IHCA with their respective survival rates. Further, to investigate whether the ETs recognised these causes during ALS. We propose a concept called "rate of recognition" (ROR).

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2. Materials and methods

2.1. Structure, material and localisation

This was a prospective observational study conducted at the St. Olav University Hospital (SOH) in Norway between January 2009 and August 2013. SOH is a 1000-bed tertiary care hospital with a regional population of 700,000. We included patients >18 years of age who received chest compressions and/or defibrillation by the ET or ward personnel. Patients were excluded if the arrests occurred as a consequence of invasive cardiac procedures, anaesthesia or surgery. Patients undergoing CPR at the time of arrival at the emergency department were defined as out-of-hospital cardiac arrest (OHCA) and not included.

The local ward personnel are routinely trained in basic CPR and the use of an automatic electronic defibrillator (AED) with a focus on immediate and high quality CPR until the arrival of the ET. The ET includes one resident in anaesthesiology, one resident in cardiology and one nurse anaesthetist with resuscitation equipment. All clinical data from patients with a written informed consent (patients or next of kin) were considered for detailed investigation.

Clinical information and defibrillation files from some of the patients were also included in our previously published papers on clinical state transitions during ALS.[13] The study is registered at clinicatrials.gov (NCT00920244). The Regional committee for medical and health research ethics in central Norway approved the study: REK 4.2008.2402, ref. no: 2009/1275.

2.2. Data collection

All relevant clinical data were extracted from the patients' records, including data from biochemical and medical imaging results, emergency-team records and Utstein style templates. Regarding the aetiology investigation, the primary variables of interest were: cause of hospital admission; cause of CA suspected by the ET; cause identified retrospectively by the authors; and whether the cause was recognised by the ET.

2.3. Aetiology analysis

In this study we defined "aetiology" as the critical underlying condition (e.g. sepsis). We defined the immediate clinical condition which was critical to the patients' haemodynamic or respiratory situation, as the "cause" or "direct corresponding cause" (e.g. hypovolaemia in the case of sepsis). To ease the interpretation, we expressed these two concepts combined as "causes".

For each episode of IHCA, a presentation of all clinical information regarding the patient, the CA episode and the post CA course was thoroughly examined by an "aetiology study group" consisting of anaesthesiologists, cardiologists and one pathologist. No additional diagnostic measures, including autopsies, were performed for study purposes.

The causes were categorised as *cardiac*, *4H4T*, *other* (including sepsis, aortic dissection, ruptured aortic aneurysm, gastrointestinal bleeding, cerebral haemorrhage/thrombosis, unspecific cancer deterioration, and anaphylactic reactions), or *unknown*. Septic shock and bleeding conditions were also included in the *hypovolaemia* subcategory within the *4H4Ts*' if symptoms were dominated by hypovolaemic shock. The total number of tamponade cardiac episodes was included in the *4H4Ts* and the tamponade cardiac episodes triggered by a cardiac cause, e.g. myocardial infarction, were included in the *cardiac* category as well. Combined critical conditions of pulmonary origin, i.e. hypercapnic/hypoxic conditions were included in the hypoxic subcategory within the *4H4Ts*'.

To determine whether ETs had correctly identified a cause, this cause had to be identified with some degree of certainty. Therefore, the causes identified through the aetiology investigation were further categorised as *reliable* or *unknown* based on the presence or absence of objective diagnostic assessments before, during or after the episodes of CA. We defined "objective diagnostic assessments" as relevant diagnostic measures clearly indicating a certain cause or excluding other potential causes. One example being clinically suspected pulmonary embolus confirmed by chest computer tomography. Another being myocardial infarction confirmed by a positive electrocardiogram (ECG) and myocardial enzyme release. Our clinical suspicion alone was not sufficient to state a certain diagnosis. Two examples from the cohort are given: a patient experienced exacerbation of her chronic obstructive pulmonary disease (COPD) until the cessation of spontaneous ventilation with ensuing hypercapnia, hypoxia, acidosis, unconsciousness and pulseless electrical activity (PEA). This patient experienced an observed arrest with immediately performed basic life support (BLS) by ward personnel. As the ET arrived, they continued ALS until return of spontaneous circulation (ROSC) was achieved shortly thereafter. A pulmonary/hypoxic cause was suspected by the ET based on clinical information about COPD with exacerbation. The patient achieved a complete recovery after supportive ventilatory measures. No additionally diagnostic assessments suggested any other potential causes, such as pneumothorax, myocardial ischaemia, septic shock, acute bleeding conditions or significant electrolyte disturbances (other than those arising from respiratory depression). The cause of arrest was defined as *hypoxia*, the degree of certainty was defined as *reliable* and the cause was *recognised* by the ET. In another patient the ALS efforts were unsuccessful and the autopsy demonstrated aortic dissection. The pre-arrest clinical data was susceptive of septic shock as the patient was hypotensive, hypoxic and anxious. The cause of arrest was defined as *aortic dissection*, the degree of certainty was defined as *reliable* and the cause was *not recognised* by the ET.

2.4. Data analysis and statistical methods

Data were analysed by applying the software Microsoft Excel (Microsoft Corporation, Redmond, WA, USA) and STATA/IC 13.1 (StataCorp LP, Texas 77845, USA). The cause-specific incidences were calculated per 1000 beds per year.

2.5. Missing data

If aetiology considerations, diagnostic measures or targeted therapeutic measures performed by the ETs were not clearly documented in the records or in other information sources described above, we were unable to decide if the ETs had recognised a correct cause. We did not perform interviews with ET participants to clarify whether they suspected a certain cause.

3. Results

3.1. Patients

A total of 306 patients were involved in 323 episodes of CA yielding an incidence rate of 72 episodes per 1000 beds per year. The details of patient inclusion are demonstrated in Fig. 1. The demographic data and reasons for hospital admission are presented in Table 1. Overall 71 patients (25%) survived to hospital discharge: 38 of 72 patients with initial VF/VT (53%), 18 of 138 patients with initial PEA (13%) and 12 of 69 patients with initial asystole (17%) as the first documented rhythm (Table 2). For three survivors there were missing data about the first rhythm.