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Clinical paper

The impact of global hemodynamics, oxygen and carbon dioxide on epileptiform EEG activity in comatose survivors of out-of-hospital cardiac arrest

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ABSTRACT

Aim: To study the association between global hemodynamics, blood gases, epileptiform EEG activity and survival after out-of-hospital CA (OHCA).

Methods: We retrospectively analyzed 195 comatose post-CA patients. At least one EEG recording per patient was evaluated to diagnose epileptiform EEG activity. Refractory epileptiform EEG activity was defined as persisting epileptic activity on EEG despite the use of 2 or more anti-epileptics. The time weighted average mean arterial pressure 48 h (TWA-MAP48), the percentage of time with a MAP below 65 and above 85 mmHg and the percentage of time with normoxia, hypoxia (<70 mmHg), hyperoxia (>150 mmHg), normocapnia, hypocapnia (<35 mmHg) and hypercapnia (>45 mmHg) were calculated.

Results: We observed epileptiform EEG activity in 57 patients (29%). A shockable rhythm was associated with a decreased likelihood of epileptic activity on the EEG (OR: 0.41, 95%CI 0.22–0.79). We did not identify an association between the TWA-MAP48, the percentage of time with MAP below 65 mmHg or above 85 mmHg, blood gas variables and the risk of post-CA epileptiform EEG activity. The presence of epileptiform activity decreased the likelihood of survival independently (OR: 0.10, 95% CI: 0.04–0.24). Interestingly, survival rates of patients in whom the epileptiform EEG resolved (n = 20), were similar compared to patients without epileptiform activity on EEG (60% vs 67%, p = 0.617). Other independent predictors of survival were presence of basic life support (BLS) (OR: 5.08, 95% CI 1.98–13.98), presence of a shockable rhythm (OR: 7.03, 95% CI: 3.18–16.55), average PaO₂ (OR = 0.93, CI 95% 0.90–0.96) and % time MAP < 65 mmHg (OR: 0.96, CI 95% 0.94–0.98).

Conclusion: Epileptiform EEG activity in post-CA patients is independently and inversely associated with survival and this effect is mainly driven by patients in whom this pattern is refractory over time despite treatment with anti-epileptic drugs. We did not identify an association between hemodynamic factors, blood gas variables and epileptiform EEG activity after CA, although both hypotension, hypoxia and epileptic EEG activity were predictors of survival.

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Introduction

Seizures are common in comatose post-cardiac arrest (CA) patients with an estimated incidence of 12–30% [1,2]. Multiple

observational studies have shown an association between the presence of seizures and worse outcome in post-CA patients [2,3]. Seizures may cause further hypoxia-induced brain damage by increasing cerebral oxygen consumption or they may just be a marker of more extended brain damage after initial resuscitation. However, the pathogenesis and the risk factors that are associated with seizures in post-CA patients remain unclear. From a clinical point of view, it is striking that patients who seem to be

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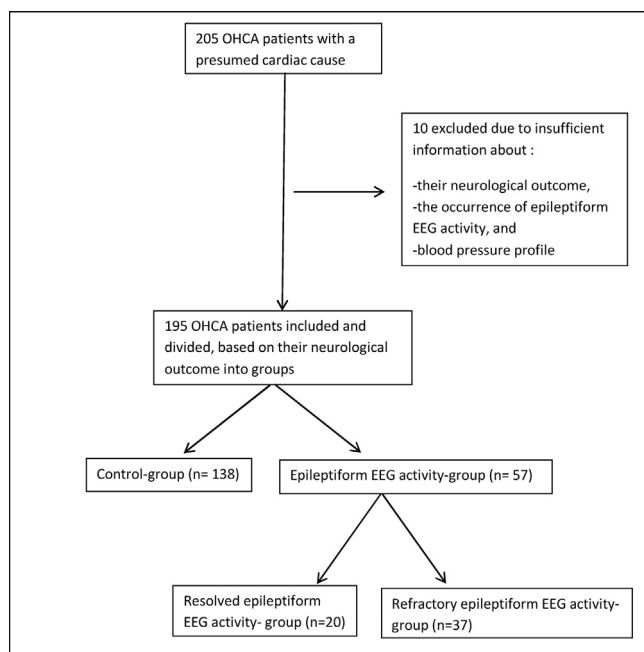


Fig. 1. Consort diagram of the patient flow: 195 comatose patients admitted to the University Hospitals Leuven and Ziekenhuis Oost-Limburg between 2010 and 2015 after being successfully resuscitated of an out of hospital CA with presumed cardiac cause were retrospectively enrolled in the database. Ten patients were excluded from the study because of insufficient information about their neurological outcome, the occurrence of epileptiform EEG activity and blood pressure profile. The patients were, based on EEG data, divided in 2 groups: the ‘control- group’, and the ‘epileptiform EEG activity-group’. The latter could be further divided in the resolved epileptiform activity group and the refractory epileptiform EEG activity group.

resuscitated well with early resumption of spontaneous contractions (ROSC) after adequate basic life support (BLS) may sometimes unexpectedly develop seizures. This suggests that other pathophysiological factors contribute to the pathogenesis of post-CA seizures. Previous studies showed an association between higher mean arterial pressures (MAP) and improved outcome in post-CA patients [4]. It has been shown that many post-CA patients have a right-shifted cerebral autoregulation [5,6]. These patients might profit from higher MAP's in order to create an adequate cerebral perfusion and avoid further hypoxia-induced damage of the cerebral penumbra. Based on the association between seizures and outcome and between MAP and outcome, we hypothesized that cerebral hypoperfusion during ICU stay might be associated with an increased risk of seizures, thereby increasing oxygen consumption and further driving hypoxia-induced brain damage. Therefore, this study aims to identify the risk factors for post-CA epileptiform EEG activity and investigates the association between hemodynamics, blood gases, epileptiform EEG activity and outcome after out of hospital CA.

Materials and methods

Study population

To be included in the study, patients had to be successfully resuscitated after an OHCA of presumed cardiac cause and had to be comatose upon arrival in the emergency department. Patients were included in the University Hospitals Leuven and Ziekenhuis Oost-Limburg between 2010 and 2015. Exclusion criteria included non-cardiac cause of the arrest such as hypoxic and traumatic CA. Ten patients were excluded from the study because of insufficient information about their neurological outcome, the occurrence of epileptiform EEG activity and blood pressure profile (see consort diagram Fig. 1). Baseline demographics and clinical characteristics,

blood tests, resuscitation characteristics, and survival, based on the patient records were included in the database. The study protocol was approved by the local medical ethics committee of both study sites. In total 195 patients were included.

Patient groups and EEG's

The patients were divided in 2 groups. The ‘control- group’, consisted of patients who did not show epileptiform EEG activity during their hospitalization. The ‘epileptiform EEG activity-group’ consisted of patients in whom epileptiform activity on an electroencephalogram (EEG) was revealed. EEG's were performed on clinical indication (clinical suspicion of epileptiform EEG activity, persisting coma after stopping sedation) and interpreted by neurophysiologists who were not involved in the ICU treatment of these patients. The first EEG was taken on day 0 in 6% of the patients, on day 1 in 15% of the patients, on day 2 in 28% of the patients (13%), on day 3 in 17% of the patients, on day 4 in 8% of the patients, on day 5 in 8% of the patients and after day 5 in 15% of the patients. A repeat EEG was performed systematically after starting anti-epileptic drugs in patients with epileptiform EEG activity. The epileptiform activity-group was further divided in a ‘resolved epileptiform EEG activity’ and a ‘refractory epileptiform EEG activity’-group. Non-refractory epileptiform EEG activity was defined as epileptic activity which was no longer demonstrated on EEG following treatment with anticonvulsive agents. Refractory epileptiform EEG activity was defined as persisting epileptiform activity on EEG despite the use of 2 or more anticonvulsive agents and increased dose of sedation.

Hemodynamic parameters

During the first 48 h after ICU admission, MAP was recorded every minute in UZ Leuven and every two seconds in ZOL Genk. Values close to 0 mmHg (leveling of the transducer) and close to atmospheric pressure (blood gas sampling) were not included in the analysis. On the basis of this blood pressure profile, the time weighted average (TWA) of the first 48 h (TWA-MAP48), the fraction of time in which a patient was exposed to MAP's below 65 mmHg during the first 48 h (time% MAP < 65 mmHg) and the fraction of time in which a patient showed MAP's above 85 mmHg during the first 48 h (time% MAP > 85 mmHg) were calculated. The TWA is a value for the average MAP which takes into account the portion of time that a patient shows a specific MAP value. This TWA equals, therefore, the sum of the portion of each time period (as a decimal), multiplied by value of the blood pressure during this time interval.

General management

All 195 patients were treated according to the previously published protocol [7]. The patients were intubated, mechanically ventilated and sedated using intravenous administration of propofol and remifentanyl. An urgent coronary angiography was performed in all patients unless a clear non-cardiac cause of death was identified. Hypothermia was induced by cold saline (4 °C – 30 ml/kg) and further maintained mechanically by endovascular cooling (Icy-catheter, CoolGard® 3000, Alsius, Irvine, CA, USA) or surface cooling (ArcticGel™ pads, Arctic Sun® 5000, Medivance, Louisville, Colorado, USA) devices for 24 h to a target temperature of 33 °C. In case of shivering during therapeutic hypothermia, cisatracurium was administered. The radial artery was cannulated for invasive blood sampling and monitoring of the MAP. In order to maintain the MAP of >65 mmHg as prescribed by the guidelines, noradrenaline (norepinephrine) was titrated. When cardiac index was below 2.0 l/min/m, dobutamine was infused and/or intra-aortic balloon pump was inserted. Blood gases were taken regularly to

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