Invited lecture/Reflection

Neuroprognostication after Cardiac Arrest

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Abstract:
Survival of patients with out-of-hospital cardiac arrest (OHCA) is still very low. After the return of spontaneous circulation (ROSC), survivors are admitted to the intensive care unit. They can be conscious or comatose. Conscious survivors of cardiac arrest generally have a good prognosis. In comatose patients, prognosis is better in patients with shockable rhythm (ventricular tachycradia or ventricular fibrillation) as the initial rhythm at the arrival of Emergency medical team.

In comatose patients we try to predict the neurological outcome with everyday clinical examination, a neuron specific enolase (NSE), comuter tomography (CT) scan or magnetic resonance imaging (MRI) of the brain, electroencephalogram (EEG) and somoatosensoric evoked potentials (SSEP). Neurological outcome is presented according to Glasgow-Pittsburgh Cerebral Performance Category Scale. Certain proportion of comatose patients may regain consciousness even after their discharge from the intensive care unit (ICU).

Keywords: Out-of-hospital cardiac arrest; Comatose survivors; Postresuscitation brain damage; Neuroprognostication

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1. Introduction

Sudden out-of-hospital cardiac arrest (OHCA) remains the leading cause of death in developed countries. Incidence varies from 50 - 100 per 100.000 inhabitants all over the world (Tadel et al, 1998). Survival is still very low, 1-22% . It is better in small towns with low traffic and no skyscrapers, where the access to the patient is quick. Prognosis is also better in patients with shockable initial rhythm, ventricular tachycardia (VT) or ventricular fibrillation (VF), and worse with non-shockable initial rhythm - pulseless electrical activity (PEA) or asystole. In many cases asystole is a secondary rhythm after non-resuscitated ventriculer fibrillation, after a few minutes due to acidosis and hypoxia.

Following initial cardiopulmonary resuscitation, reestablishment of spontaneous circulation (ROSC) is typically achieved in 40 to 60% of patients who are subsequently transported to the hospital. There is an increasing number of patients admitted to our ICU each year, starting from 25-35 per year until 2002, to maximum 90 patients per year until now (**Figure 1**). From 1995 until 2021 we admitted 1352 patients after primary OHCA (Tadel-Kocjancic et al., 2022).

Because of typical delays in prehospital "chain of survival", a great majority of patients remain comatose despite ROSC and require intensive post-resuscitation care (Nolan et al., 2021) Introduction of hypothermia after the publication of landmark clinical trials in 2002 undoubtedly revolutionized post-resuscitation treatment (Hypothermia after Cardiac Arrest Study Group, 2002). Such comprehensive post-resuscitation care has been shown to significantly improve survival with good neurological outcome compared to historical controls. Conscious patients (the ones who re-gained consciousness after ROSC) have excellent prognosis.

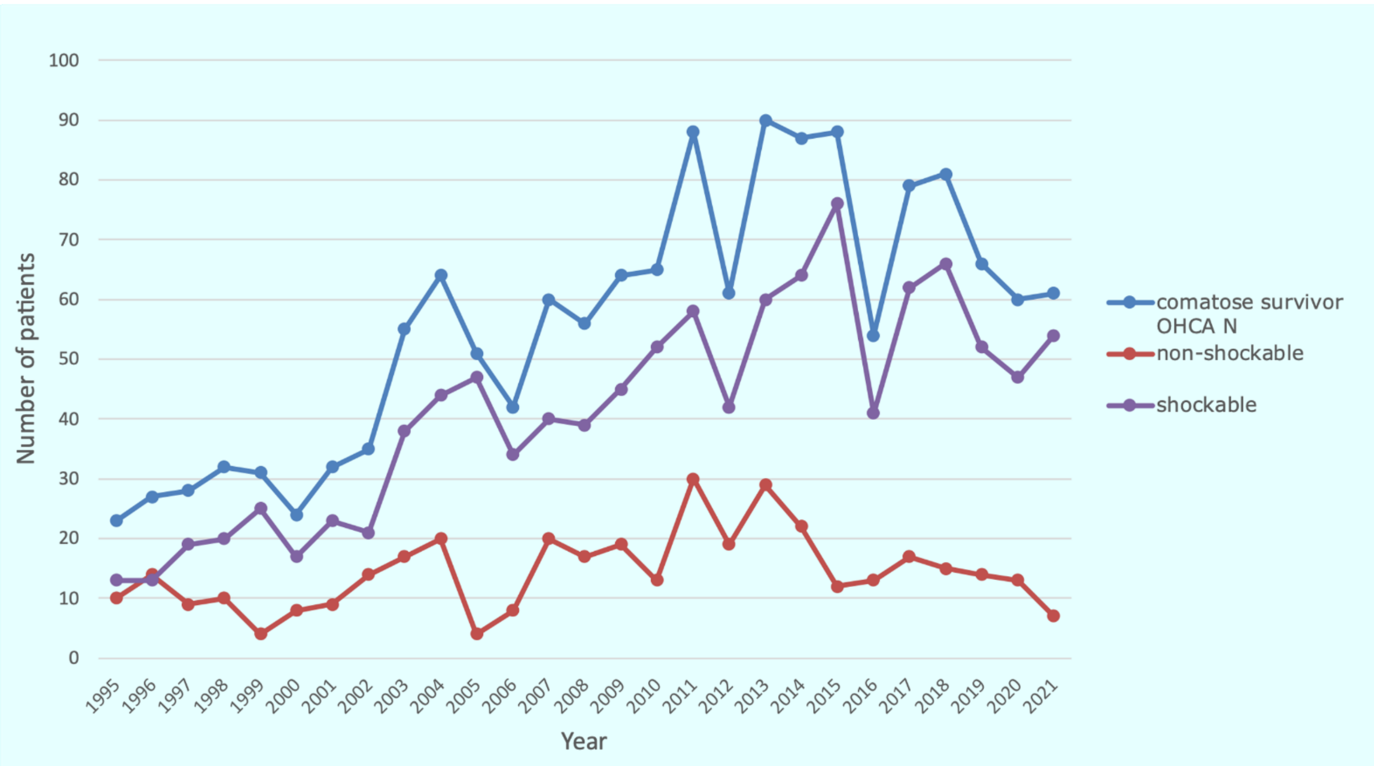

Figure 1. Number of comatose survivors of OHCA admitted to ICU after ROSC.

2. Brain damage after cardiac arrest

During cardiac arrest there is no blood flow through the body resulting in ischemic damage to the organs. Brain seems to be the most vulnerable, and patients often suffer an irreparable brain damage after cardiac arrest. If the patient regains consciousness after ROSC, there is no or very little brain damage. About 70% of patients remain comatose, which means that brain damage is very likely. After ROSC there is also post-resuscitation brain damage which we try to limit with therapeutic hypothermia or normothermia for 48 hours.

At first, it is hard to assess consciousness due to the sedatives, analgesics and/or muscle relaxants the patients receive during cardiopulmonary resuscitation. Neuroprognostication comes later. After all that we classify patients into 5 groups according to Glasgow-Pittsburgh Cerebral Performance Category Scale (CPC) (**Table 1**). Standard definitions are: CPC 1 to 2 - favorable and 3 to 5 - poor neurologic outcome.

Table 1. Glasgow-Pittsburgh Cerebral Performance Category Scale (Safar et. al.,1986).

Note: If patient is anesthetized, paralyzed, or intubated, use "as is" clinical condition to calculate scores.
CPC 1. Good cerebral performance: conscious, alert, able to work, might have mild neurologic or psychologic deficit.
CPC 2. Moderate cerebral disability: conscious, sufficient cerebral function for independent activities of daily life. Able to work in sheltered environment.
CPC 3. Severe cerebral disability: conscious, dependent on others for daily support because of impaired brain function. Ranges from ambulatory state to severe dementia or paralysis.
CPC 4. Coma or vegetative state: any degree of coma without the presence of all brain death criteria. Unawareness, even if appears awake (vegetative state) without interaction with environment; may have spontaneous eye opening and sleep/awake cycles. Cerebral unresponsiveness.
CPC 5. Brain death: apnea, areflexia, EEG silence, etc.

For evaluation of comatose patients we use everyday clinical neurological examination, levels of NSE 72 hours after ROSC, EEG recordings on day 3, SSEP and CT of the brain (Henson et al., 2022). Daily clinical neurological examination is performed. We must be careful to exclude the influence of sedatives, muscle relaxants on consciousness and reflexes. Signs of poor neurological outcome are absent or extensor motor response to pain at 72 h or later after ROSC, bilaterally absent pupillary light reflex at ≥ 72 h from ROSC, bilaterally absent corneal reflex at 72 h after ROSC, presence of an early (≤ 48 h) post-anoxic status myoclonus. NSE levels are measured 72 hours after ROSC. High levels (mostly more then 60) mean a bad prognosis. On day 3-5 we record EEG. We then divide EEG recordings into 3 groups (discretion of the neurophysiologist who interpret them): very malignant, malignant, or benign recording. The presence or absence of SSEP is noted. Absence of somatosensoric evoked cortical N20 potentials means poor neurological outcome, but to correctly interpret these findings, injuries to the cervical spinal cord must be excluded.

Neuroimaging is also used for neuroprognostication. MRI or CT of the head are performed, mostly CT in our hospital. The findings are then interpreted by a radiologist. Generalized brain edema, where we cannot distinguish between white and grey matter, extensive diffusic restrition on MRI or extensive lesions mean bad prognosis, but focal lesions have no significant clinical importance.

3. Delayed awakening of patients after OHCA

Despite advanced neuroprognostication using NSE, EEG and brain imaging, prediction of neurological outcome in comatose survivors of OHCA remains challenging and early discontinuation of post-resuscitation treatment may be harmful for patients with delayed awakening. In our study (Tadel-Kocjancic et al., 2022) we find that in about 20% of patients in CPC 3 or 4 there is neurological improvement later, so it is important that we do not stop treatment too early.

4. Conclusion

Survival of patiens after OHCA is still very low. Conscious survivors have a good prognosis, but shockable initial rhythm (VT/VF) means a better prognosis for comatose survivors. Intensive hospital treatment improves prognosis in patients with shockable rhythm, but not in patients with non-shockable rhythm.

In the last years we have been trying to predict neurological outcome in comatose patients with neuroprognostication. We have been using everyday clinical examination, NSE, neuroimaging and neurophysiologic tests. We hope that with all this data our decision whether to continue or to stop treatment will be easier.

Conflicts of Interest: The author declares no conflict of interest.

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