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Good Outcome Despite Prolonged Absence of EEG Reactivity Following Myoclonic Status Epilepticus in a Patient after Hypothermia Post Cardiac Arrest

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Abstract

Prognosis of neurological outcome after cardiac arrest, post hypothermia has been an ever developing issue. Absence of EEG reactivity has been one of the indicators that have been suggested to be promising in such a scenario. We present a case of a patient who underwent hypothermia post-cardiac arrest and had absent EEG reactivity even 3 weeks after cardiac arrest, but had good neurological outcome at discharge.

Introduction

Our consensus on prognosis after cardiac arrest is based upon data from pre-HACA era and include absence of pupillary or corneal reflex on day 3, bilateral extensor posturing or absent motor responses on day 3, clinical myoclonic status epilepticus within 24 hours, serum neuron-spicific enolase greater than 33 ug/L on days 1-3 and SSEPs showing bilateral absent N20 responses [1]. However, based upon studies done after hypothermia-era we have data to support that motor examination [2,3], serum neuron-specific-enolase [4], early myoclonus [2] and pupillary or corneal [2] cannot provide fool-proof prognostication in HACA patients. The same studies have provided support for absence of EEG reactivity as an additional marker for poor prognosis [2]. We present a case of a patient who underwent hypothermia after an out of hospital cardiac arrest, went into myoclonic status epilepticus and had absence of EEG reactivity, but had good neurological outcome.

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Case Presentation

A 67 year old man collapsed inside the car was found to be in cardiac arrest when the EMS arrives and was defibrillated seven times at the scene, during transport, and in the ED with return of spontaneous circulation (ROSC) after 33 minutes of resuscitation. He underwent therapeutic hypothermia, initiated within 6 hours after ROSC and controlled rewarming was done after 24 hours. The patient had reactive pupils immediately post resuscitation. On day 6, after discontinuation of sedation, the patient remained unresponsive EEG showed background suppression with generalized slowing but no epileptiform activity. SSEPs showed bilateral cortical responses. On day 7, myoclonic jerking of the face and arms were noted intermittently. Keppra was started without any improvement. Myoclonus increased in frequency, intermittently responding to lorazepam. Repeat EEG showed rhythmic polyspike and wave consistent with status epilepticus, accompanied with facial myoclonus. MRI of the brain showed only subtle T2/FLAIR hyperintensity along caudate and bifrontal cortical ribbon.

The patient was transferred to the neuro-ICU and EEG monitoring was started for burst suppression therapy with midazolam which was switched to pentobarbital infusion Pentobarbital was successfully weaned after 15 days and AEDwas were tapered.

The patient remained comatose 3 weeks after resolution of status epilepticus with subsequent EEG showing no reactivity to auditory or tactile stimuli. On day 60, repeat EEG showed reappearance of posterior dominant rhythm with reactivity and presence of sleep activity during sleep state. Eye opening was noted with verbal stimulation but no regard and no volitional movement was noted. On day 64, the patient was noted to track and answer yes and no with head nodding. Next day, he was consistently following commands and was able to mouth words. The patient was discharged on day 70 to sub-acute rehabilitation facility, with ability to communicate and move his extremities purposefully.

Discussion

Absence of patient's spontaneous circulation for 33 minutes was sufficient enough to cause diffuse brain injury. The patient developed myoclonic status epilepticus after 7 days of the sentinel event In addition to being in status for over 2 weeks thereafter, patient's EEG did not show any reactivity to auditory or tactile stimuli.

Patient's myoclonic status (even though not early) and absent EEG reactivity predicted poor prognosis on one hand, the presence of bilateral N20s on SSEP clouded our judgment regarding prognosis on the other. However, patient's significant recovery at the time of discharge showed that further studies are indicated to find better prognostic signs in anoxic brain injury patients who undergo therapeutic hypothermia. Recent studies have already successfully disputed conventional markers of prognosis in the hypothermia-era [2,3]. Inclusion of EEG reactivity- even as late as 3 weeks after cardiac arrest as shown in our case- as a marker of poor prognosis needs further studies and should not be used as a marker with only a few studies on board [2,3].

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