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CASE REPORT

Recovery from near drowning and postanoxic status epilepticus with controlled hypothermia

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ABSTRACT

A diver was resuscitated after cardiac arrest due to near drowning and was hypothermic on hospital arrival. During rewarming, status epilepticus occurred, previously identified as a predictor of poor outcome. The seizures responded well to treatment with antiepileptic drugs and controlled hypothermia. After six weeks, the patient had completely recovered. This case supports the hypothesis that hypothermia offers neuroprotection, even in the presence of status epilepticus. We recommend that near-drowning victims who are comatose after resuscitation for cardiac arrest be treated with controlled mild hypothermia for 12 to 24 hours.

KEYWORDS

Brain hypoxia, induced hypothermia, near drowning, resuscitation, status epilepticus

INTRODUCTION

Controlled hypothermia is recommended in the resuscitation guidelines created by the International Liaison Committee on Resuscitation (ILCOR) to limit neurological damage in patients resuscitated for out-of-hospital cardiac arrest.[1] Although evidence about temperature management in resuscitated near-drowning victims is lacking, it has been recommended to treat these patients in a similar way.[2] Nevertheless, reports about the results of controlled hypothermia in near-drowning victims are scarce.[3-6] In this case report, we present a near-drowning victim who recovered completely after treatment with controlled hypothermia, despite postanoxic status epilepticus, which has previously been identified as a predictor of poor outcome.

CASE REPORT

A 44-year-old male diver lost his mouthpiece and was found pulseless with asystolic heart activity 18 minutes later. Ten minutes after resuscitation was started, spontaneous circulation returned. On hospital arrival, the Glasgow Coma Score was 3 and the body temperature 30.1 °C. The patient was mechanically ventilated and haemodynamically stable under sedation with propofol. The pupils were dilated and not reactive to light, while corneal and oculocephalic reflexes were absent. Laboratory results showed lactic acidosis (pH 7.01, lactate 20.3 mmol/l). Since the clinical situation was stable and the core temperature was below 32 °C, the patient was allowed to rewarm according to international guidelines.[1] However, the temperature accidentally rose to 38 °C and recurrent tonic-clonic seizures occurred, compatible with status epilepticus.[7] Controlled hypothermia with a target of 33 °C was applied for 24 hours and the seizures were treated with valproic acid and levetiracetam. Three days later, somatosensory evoked potentials revealed a bilateral intact N20 response and the electroencephalogram showed a slow background pattern without ictal activity. A week later, the patient regained consciousness and six weeks after the accident, he had completely recovered. Neuropsychological assessment six months after the accident showed no deficits.

DISCUSSION

Controlled hypothermia limits neurological damage in resuscitated patients and might therefore also be beneficial in resuscitated near-drowning victims.[2] Successful use of controlled hypothermia in these patients has been reported previously.[3-6] It is hypothesised that controlled hypothermia not only decreases cerebral metabolism, but also limits

the effects of ischaemia and reperfusion on the brain. Animal studies have shown that controlled hypothermia postpones ischaemic depolarisation and inhibits the increase in excitatory neurotransmitters such as glutamate and dopamine.[8,9] In addition, controlled hypothermia inhibits the metabolism of arachidonic acid, limiting the production of cell membrane damaging metabolites such as prostaglandins and eicosanoids.[10] Finally, controlled hypothermia has an anti-inflammatory effect: it inhibits the production of cytokines and adhesion molecules, thereby limiting polymorphonuclear cell infiltration and oxygen radical production.[11]

Controlled hypothermia might have added to the favourable outcome in our patient. To our knowledge, this is the first report of a near-drowning victim who recovered completely despite postanoxic status epilepticus, which previously has been identified as an independent predictor of poor neurological outcome when occurring within 24 hours after cardiopulmonary arrest.[12] In addition, a recent paper described two patients with postanoxic status epilepticus after resuscitation for primary cardiac arrest, who experienced a favourable outcome after treatment with controlled hypothermia.[13]

Whether the fever occurring during rewarming precipitated the occurrence of status epilepticus in our patient remains speculative. Although it is well known that fever can induce seizures in animals and children, this has never been demonstrated in adults. Therefore, it seems unlikely that fever caused epilepsy in our patient. However, fever has been described as a phenomenon accompanying the presentation of status epilepticus.[14]

A limitation of the current case report is the fact that status epilepticus was not confirmed electrographically before treatment with anticonvulsive agents. The value of continuous amplitude-integrated electroencephalography in patients with postanoxic status epilepticus has been described previously.[15-17] In a recent study, 26 of 95 resuscitated patients treated with hypothermia experienced postanoxic status epilepticus. The outcome of these patients was related to the way status epilepticus developed: two of ten patients with status epilepticus developing from a continuous background regained consciousness, whereas none of 16 patients with status epilepticus developing from suppression burst background did.[15] In the two patients who regained consciousness, status epilepticus occurred after rewarming, just as in our patient.

In summary, the current case supports the hypothesis that controlled hypothermia may offer neuroprotection in patients with postanoxic encephalopathy, even in the presence of status epilepticus. We recommend that near-drowning victims who are comatose after resuscitation for cardiac arrest be treated with controlled mild hypothermia for 12 to 24 hours.

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