

CASE REPORTS

Neurological outcome prediction in a cardiorespiratory arrest survivor**W. C. Goh^{1*}, P. D. Heath², S. J. Ellis³ and P. A. Oakley⁴**

Departments of ¹Anaesthesia and Intensive Care, City General, ²Clinical Neurophysiology, ³Neurology, ⁴Trauma Research, Royal Infirmary, North Staffordshire Hospital, Princes Road, Hartshill, Stoke-on-Trent ST4 7LN, UK

*Corresponding author

Outcome prediction of neurological recovery in an unconscious survivor of cardiorespiratory arrest is difficult and uncertain. We describe the case of a 25-yr-old post-arrest survivor who made a remarkable neurological improvement despite a seemingly hopeless prognosis. Conventional clinical and neurophysiological assessments need to be interpreted with care in the presence of uncontrolled seizure activity and sedative medications. The measurement of biochemical markers in the serum and cerebrospinal fluid may be useful in helping the clinician to arrive at a more accurate neurological outcome prediction.

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With the advent of emergency medicine and improvements in the provision of emergency medical services, the number of patients who survive a cardiorespiratory arrest has increased. However, many of these survivors never regain consciousness¹ and progress to a persistent vegetative state.² Considerable research has been carried out to identify those comatose patients who will recover sufficiently to live a meaningful life.³ An accurate prediction of neurological outcome not only has ethical and legal implications but is also in the interest of the patient and his or her family. The ability to predict very poor outcome would relieve pressure on the finite resources of intensive care. At present, such outcome predictions are based on clinical history and physical examination, electrophysiological findings, neuroimaging tests and levels of biochemical markers in the serum and cerebrospinal fluid (CSF). However, considerable uncertainty remains. We present a case of a 25-yr-old survivor of cardiorespiratory arrest who made a remarkable neurological recovery despite a seemingly hopeless prognosis. This is followed by a discussion of the various factors that led to the initial decision to withdraw treatment. We will also explore other factors and additional tests that might have helped us to predict neurological outcome more accurately.

Case report

A 25-yr-old male with a history of poorly controlled asthma collapsed at home in the late afternoon, having complained of shortness of breath since the day before. The collapse was witnessed by his father, but it was unlikely that effective cardiopulmonary resuscitation (CPR) had been carried out before the arrival of the ambulance crew. When the paramedics arrived, 6 min after the emergency services had been contacted, there was no respiratory effort and no palpable pulse. The ECG monitor showed a heart rate of 24 beats min⁻¹. CPR was commenced and the patient's trachea was intubated. After a further 3 min, the carotid pulse became palpable, increasing to a rate of 107 beats min⁻¹. The patient started to breathe but remained unconscious, with a Glasgow coma score (GCS) of 3. An oxygen saturation of 98% was achieved at the scene with assisted ventilation and the patient was transferred to the accident and emergency department.

On admission to the accident and emergency department, the patient was making spontaneous respiratory effort and his oxygen saturation was 100%. The heart rate was 120 beats min⁻¹, with an arterial pressure of 190/120 mm Hg. The pupils were dilated but reactive to light. The patient was initially unresponsive, and within 5 min he

adopted a decorticate posture. He was sedated with propofol, given atracurium and transferred to the intensive care unit (ICU) for further management.

In the ICU, the patient remained haemodynamically stable at most times, occasionally requiring small amounts of norepinephrine to keep his mean arterial pressure above 90 mm Hg. Bronchospasm was present but not severe, and improved with salbutamol and ipratropium nebulisers, hydrocortisone and aminophylline. The following day, the patient displayed frequent myoclonic jerks, involving the head and all four limbs. The seizures failed to respond to phenytoin, magnesium and repeated doses of clonazepam. Later that day, a CT scan of the head was performed and was normal. The serum theophylline level was within the therapeutic range. An EEG recording made after withdrawing sedation showed generalized status epilepticus. At this time the prognosis was thought to be very poor. However, it was considered inappropriate to make a palliative care decision in the face of status epilepticus that would mask his potential conscious level. The plan was to control the fits with more anticonvulsants and then reassess his conscious state after stopping sedation. On the basis of repeated EEGs, the seizures proved impossible to control with therapeutic doses of phenytoin, midazolam, clonazepam, sodium valproate and magnesium sulphate. A thiopental infusion was added and burst suppression was achieved. However, after the thiopental infusion was stopped, the patient remained comatose and the seizures recurred. A subsequent EEG showed return of epileptic activity and the appearance of generalized alpha frequency activity. Whilst potentially drug induced, this recording suggested the possibility of alpha coma and serious pontine dysfunction. In view of his uncontrolled seizure activity and poor neurological state, it was accepted that the patient had suffered severe hypoxic brain injury and that there was no likelihood of him returning to an independent existence. A palliative care decision was agreed with the family. Since the patient was breathing spontaneously, his trachea was extubated and he was transferred to the general ward on day 6.

In the general ward, the patient showed progressive improvement in his neurological state. Initially, his GCS was 3/15 but this had improved to 10/15 by day 16. At this time he was able to answer questions appropriately by nodding his head and to pronounce his name. He still had generalized muscular weakness, but was able to move both arms and legs according to command. He could maintain balance in a sitting position. In view of this neurological progress, the palliative care decision was reversed. At day 34, he was orientated to date and time. He could repeat short sentences and count backwards from 20 to 1. He had no contractures and demonstrated almost full power in both arms and legs. Sensation in the limbs was normal. He could sit up from a supine position and stand up from a sitting position with assistance. He was able to swallow thick fluids. At this point, he was referred to the rehabilitation unit for further management.

Throughout his stay in the ward, the patient continued to exhibit recurrent myoclonic jerks. He was put on various anti-epileptic regimens, which included phenytoin, phenobarbital, sodium valproate, clonazepam and piracetam. An EEG recording performed on day 18 showed an improvement in cerebral activity, but prominent epileptic activity persisted and this correlated well with the myoclonic jerks. A CT scan of the head on day 19 was normal. The myoclonic jerks were more frequent and severe in amplitude when the patient attempted purposeful movements. They continued to interfere with his rehabilitation, particularly his ability to stand unsupported.

At week 19, the patient had a GCS of 15. He was orientated to time and place and was able to converse, although some dysarthria persisted. He had normal pupillary reflexes, no visual or hearing impairment, no facial asymmetry, and had normal power and sensation in his arms and legs. He was able to walk the whole length of the ward with a Zimmer frame and with assistance from two people. He could eat a normal meal, including solid food, and wash and dress himself with some assistance. He did not have urinary or bowel incontinence. His myoclonic jerks were less frequent and less severe. He was discharged home from the rehabilitation ward, with outpatient appointments for physiotherapy.

Discussion

This man suffered a severe hypoxic episode, followed by a cardiac arrest. He may have had no cerebral perfusion for up to 6 min before the ambulance crew arrived. It took a further 3 min of CPR before a pulse became palpable. Berek and colleagues⁴ have shown that there is a significant difference in the duration of anoxia between patients with favourable (mean 4.1 min) and unfavourable (mean 8 min) outcome after CPR. Three hours after the collapse, the patient was still unconscious in the accident and emergency department. The duration of postanoxic coma has been reported as another prognostic indicator. With intensive therapy, 20% of patients who are unconscious 10 min after successful CPR have normal or near-normal health after 1 yr, whereas the others have severe impairment of brain function until death.⁵ Our patient also developed decorticate rigidity and had a generalized fit. It is important to note that this patient had no history of epilepsy, and it would therefore be reasonable to attribute the cause of the fit to hypoxic cerebral damage.

In the ICU, the patient continued to have clinical and EEG evidence of epileptic activity which was impossible to control. There was no improvement in the conscious level when the anaesthetic and most of the sedative agents were discontinued (the patient was still receiving clonazepam, phenytoin and sodium valproate). Several authors have observed that persistence of unresponsive coma for 48 h after cardiac arrest is predictive of a poor outcome.^{6 7} Furthermore, the EEG recording also raised the possibility

of alpha pattern coma. Most cases of alpha coma die or survive for varying periods in the vegetative state.⁸ Kaplan and colleagues⁹ estimated the mortality rate of alpha coma after cardiorespiratory arrest to be 88%. Our patient also developed generalized myoclonic jerks. Posthypoxic myoclonus is commonly thought to lead to severe and permanent disability.¹⁰

In spite of all these poor prognostic features, the patient made good neurological recovery in the ward. It is possible that there was some on-going systemic perfusion during the collapse which was followed by electromechanical dissociation just before the arrival of the ambulance crew. This would have supplied small but vital amounts of oxygen to the brain during this critical period. The patient may have had a circulation that was difficult to detect because of the bradycardia. There was return of spontaneous respiration after resuscitation, and there was pupillary response to light in the accident and emergency department. Absence of pupillary light response and motor response to pain are associated with a poor prognosis.^{11 12} Subsequent neurological assessment of the patient in the ICU was difficult because of recurrent epilepsy, sedation and paralysis. Residual levels of anaesthetic and sedative agents after discontinuation could account for the depressed conscious level and the alpha pattern EEG, which may be seen in anaesthesia and drug-induced coma and is associated with recovery of normal consciousness.¹³

One test that may help in the assessment of potential neurological recovery is the cortical somatosensory evoked potentials (SSEP). This is a non-invasive bedside test that has been used for prognostic evaluation in cases of hypoxic coma following cardiac arrest. Absence of the short latency SSEP (N20) peak is associated with poor outcome.¹⁴⁻¹⁸ Absence or delay of the long latency (N70) peak is also an indicator of poor prognosis.¹ It is suggested that a recording of the N70 peak is more accurate in predicting individual outcome than physician review of clinical data.¹⁹ However, SSEP is difficult to record reliably in an intensive care environment. The anaesthetic and sedative agents used in patient management can themselves depress or extinguish the evoked potentials, and status epilepticus will interfere with SSEP recording.

Elevated blood concentration of neurone-specific enolase (NSE) is indicative of a poor prognosis in ischaemic brain damage.²⁰ CSF levels of brain-specific creatine kinase isoenzyme (CKBB), lactate and NSE are also found to reflect patient outcome.²⁰⁻²² CSF CKBB activity of more than 205 units per litre measured 48-72 h after cardiac arrest is associated with non-awakening.

It is interesting to note that in a follow up of 14 post-arrest patients with chronic posthypoxic myoclonus (11 of these cases are caused by an acute asthmatic attack), the myoclonus improved with time and severe neurological deficits were rare.²³ This case report demonstrates how conventional clinical and EEG assessment of neurological recovery from cerebral hypoxia after cardiac arrest follow-

ing an acute severe asthmatic attack can be misleading. Great care needs to be taken in the interpretation of both clinical and neurophysiological data in the presence of uncontrolled epilepsy and sedative medications. The decision to adopt a palliative care plan is fraught with uncertainty. Such a decision should be reviewed in the light of unexpected neurological improvement.

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