SHORT REPORT

Early myoclonic status and outcome after cardiorespiratory arrest

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Abstract

It has been suggested that early myoclonic status after cardiorespiratory arrest is an agonal event.¹ Here we describe three cases who developed early myoclonic status during a coma after cardiorespiratory arrest due to acute asthma. As consciousness improved, each patient developed Lance-Adams type multifocal myoclonus, but the eventual outcome was satisfactory. Only one patient needed assistance to walk, and all three were self caring. One patient had persistent dyscalculia. Early myoclonic status is not necessarily an agonal event, particularly when it follows arrest due to acute asthma or asphyxia.

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Neurologists are often called on to evaluate patients with brain injury after cardiorespiratory arrest and to offer advice on prognosis. The early clinical signs that may predict outcome continue to be validated in clinical trials. It is important that potential prognostic indicators are evaluated in a prospective treatment controlled setting, otherwise there is a danger that certain clinical signs may be perceived as being incompatible with survival, leading to limitation of patient care and a self fulfilling prophesy.² The Brain Resuscitation Clinical Trials I Study Group has shown that a useful prognostic indicator is the absence of a motor response to pain at 72 hours after arrest which, in a series of 262 patients after cardiac arrest, has been 100% predictive of a poor outcome.³ This finding has been confirmed in subsequent studies and, importantly, these studies emphasise the unreliability of early clinical signs in predicting outcome.⁴

Wijidicks *et al* have reported a series of 107 patients in coma after cardiorespiratory arrest and found that all patients with early myoclonic status, defined as spontaneous and touch sensitive generalised myoclonic jerks noted within 24 hours of coma onset, died.¹ They concluded that myoclonic status after cardiac arrest is an agonal event and must "strongly in future influence the decision to withdraw life

support". We report three survivors after early myoclonic status.

Case reports

CASE 1

A 23 year old man had an asystolic cardiac arrest while being transferred by car to hospital during an acute asthma attack. Cardiac output was restored within five minutes. On admission to the intensive care unit his pupils were unreactive to light and he had widespread spontaneous and stimulus sensitive myoclonic jerks. He subsequently developed generalised tonic clonic seizures which were controlled with carbamazepine, phenytoin, and sodium valproate, and propofol or thiopentone anaesthesia. His sedation was gradually withdrawn and he was extubated 22 days after arrest at which time he was able to obey complex commands and soon became fully oriented. As his functional ability improved it became obvious that he had Lance-Adams type multifocal action myoclonus which compromised his gait and manual dexterity. He attended a neurorehabilitation centre and was discharged home four months after arrest. Sixteen months later he was self caring and able to transfer unaided, walk with the assistance of one person, and was cognitively intact.

CASE 2

A 54 year old woman with a history of asthma, developed left shoulder and arm pain with breathlessness, for which she took non-steroidal anti-inflammatory analgesia. Her condition worsened and during transfer to hospital she had a ventricular fibrillation cardiac arrest. External cardiac massage and ventilation were initiated and cardiac output was restored around 15 minutes later. On admission to the intensive care unit she was noted to have jerky facial grimacing after endotracheal suction. A few hours later as her benzodiazepine sedation was withdrawn she developed spontaneous and touch sensitive myoclonus affecting the face and all four limbs. She was extubated five days after the cardiac arrest. Two days later she was obeying commands, at which point she was noted to have multifocal action myoclonus. Treatment was begun with sodium valproate and clonazepam. She continued to make a steady recovery and five months after arrest she was

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self caring, able to walk unaided, and had normal cognitive function apart from moderate dyscalculia. Myoclonic jerks were small and infrequent. Coronary angiography and ventricular tachycardia stimulation studies were normal. Subsequently, it was concluded that she had had a bronchoconstrictor reaction to the non-steroidal anti-inflammatory drugs.

CASE 3

A 19 year old man with a history of mild asthma developed acute breathlessness, followed by collapse. He was attended by a paramedical team who found no cardiac output, and intubated and ventilated him. His cardiac output was restored and he was sedated, paralysed, and ventilated. The duration of the cardiac arrest was not documented. As neuromuscular paralysis was discontinued 30 hours after the arrest he developed widespread, almost continuous spontaneous myoclonic jerks and tonic-clonic seizures. These were controlled with benzodiazepines and phenytoin. He was extubated two weeks after admission to the intensive care unit at which time he was obeying commands and mouthing words. Thereafter, voluntary movements were disrupted by Lance-Adams type multifocal myoclonus, but he made a good recovery over the ensuing years. Four years later he was walking unaided and was entirely self caring, although his speech was slightly interrupted by myoclonus. There were no cognitive difficulties and he had no more seizures. He required neither antiepileptic nor antimyoclonic medication.

Discussion

These three case reports add to the literature on survival with myoclonus after cardiorespiratory arrest and specifically address the question of the time of onset of myoclonic activity. Two of our patients developed myoclonic status within 24 hours of cardiorespiratory arrest and a third patient developed myoclonus 30 hours after arrest, just as sedation and neuromuscular paralysis were discontinued.

There are other reported cases of survival after early myoclonic status. Arnoldus and Lammers reported on a patient who developed widespread spontaneous and stimulus sensitive myoclonus four hours after cardiorespiratory arrest due to laryngeal oedema who, on regaining consciousness, had Lance-Adams type

action myoclonus, but normal cognitive function.⁵ Werhahn *et al* reported two further patients with early myoclonic status after asthmatic cardiorespiratory arrests (one of whom was aged 72), who made similar recoveries, although description of the period of coma was limited.⁶ Finally, Harper and Wilkes reported two patients with early myoclonic status after asthmatic cardiorespiratory arrests, who made full recoveries with the exception of persisting action myoclonus.⁷

These, and our cases of early myoclonic status, occurred in the setting of cardiorespiratory arrest secondary to acute respiratory difficulties, rather than primarily cardiac events, raising the possibility that metabolic changes before the arrest might limit anoxic brain damage and favour recovery.⁸ The aetiology of postanoxic coma also seems to influence the nature of the clinical deficit in those patients that do recover, and it is well recognised that the Lance-Adams syndrome of action myoclonus tends to be seen in those patients surviving arrests due to asthma or asphyxia rather than primarily cardiac events.⁶

We conclude that myoclonic status in postanoxic coma need not be an agonal event and that some patients with early myoclonic status survive to develop the Lance-Adams syndrome. This, together with the knowledge that most survivors developing the Lance-Adams syndrome have a satisfactory long term outcome,⁶ should temper decisions to withdraw life support in patients with early myoclonic status after cardiac arrest, especially when this occurs in the setting of acute asthma or asphyxia.

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