

CASE REPORT

Temporary recovery after resuscitation: delayed postanoxic encephalopathy

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SUMMARY

Delayed postanoxic encephalopathy is reported infrequently and is characterised by a lucid interval of seemingly good recovery from an anoxic insult. Days or even weeks may pass before changes in behaviour, motor responses or consciousness occur. Neurological deterioration may progress to coma, death or severe disability, whereas some patients have a second recovery period. Pathogenesis is yet to be discovered. Prognosis is reportedly poor, treatment is symptomatic and at best anecdotal. We present a case report and available literature.

BACKGROUND

It is important to recognise the delayed onset of postanoxic encephalopathy in the clinical setting of a lucid interval of seemingly good recovery from an anoxic insult. It has been reported throughout medical literature in a small number of case reports and carries a poor prognosis. Classically, there is a lucid interval after initial good recovery from an anoxic insult, upon which neurological deterioration may progress to coma, death or severe disability.

CASE PRESENTATION

A 69-year-old man was seen at the emergency department after he had been resuscitated by a neighbour. She had found him unconscious and pulseless, upon which she had initiated resuscitation. Upon arrival of the paramedics he had spontaneous circulation and was agitated. Because of vomiting and aspiration he was intubated for airway protection. His medical history reported hypertension and diabetes mellitus type 2.

At presentation at the hospital his Glasgow Coma Score was 3, with anisocoria and absent oculocephalic reflex but intact pupillary light and corneal reflexes. His circulation was stable with a blood pressure of 200/100 mm Hg and a regular pulse. Oxygenation was insufficient and required mechanical ventilation using 100% oxygen. After 24 h the patient gradually improved.

INVESTIGATIONS

Laboratory examination showed a lactic acidosis (lactic acid 5).

CT scan of the brain revealed no abnormalities, nor did an additional CT angiography.

ECG revealed ST-depression mostly over the anterior cardiac wall. This was confirmed by echocardiography, which showed no movement of part of the anterior wall. Furthermore, the ECG revealed a suspicion of Wolff-Parkinson-White (WPW) syndrome.

The troponin T levels were elevated with a maximum of 0.2 µg/l (normal value 0.0–0.1 µg/l).

We concluded an arrhythmia caused by anterior myocardial infarction or possibly a previously unknown WPW syndrome as a cause of the cardiac arrest.

OUTCOME AND FOLLOW-UP

At the intensive care unit he recovered to a maximum neurological score of opening his eyes on verbal command and flexing his legs on demand. However, movement in his arms appeared to be limited to only flexing on pain. There was no clear visual orientation and he still depended on mechanical ventilation. After 4 days, he deteriorated after a generalised tonic-clonic seizure. Prior to this he had not revealed any seizure-like activity. Therefore this was considered symptomatic upon which Valproate was initiated. EEG revealed no epileptic activity. Subsequent serial EEG recordings showed sharp-wave complexes at the vertex (figure 1), which ceased on a bolus of Midazolam. Hence treatment was intensified with intravenous Phenytoin, however without improvement. An MRI scan of the brain showed hyperintense zones on T2-weighted images compatible with cortical laminar necrosis (figure 2). After nearly 3 weeks he showed no further improvement other than opening his eyes on verbal command without goal-directed eye movements and pathological flexing of the arms on pain without movement of the legs. A second MRI of the brain displayed an increase in the previously found hyperintensities (figure 2). Despite maximal treatment the clinical situation remained poor and treatment was stopped.

DISCUSSION

The patient we presented deteriorated after initial clinical improvement from postanoxic coma. After ruling out several causes such as non-convulsive status epilepticus, basilar artery thrombosis and metabolic causes, a delayed postanoxic encephalopathy was diagnosed. As early as in 1917, a woman was described with initial full recovery after strangulation followed by a severe neurological deterioration after 5 days.¹ Autopsy revealed grossly visible cavities in striatum and caudate nucleus, but an otherwise intact brain. Several other case reports followed up to a comprehensive overview of five cases in 1962.² The lack of awareness of delayed postanoxic encephalopathy was already noted in this paper and is still reflected by the scarce subsequent reports.

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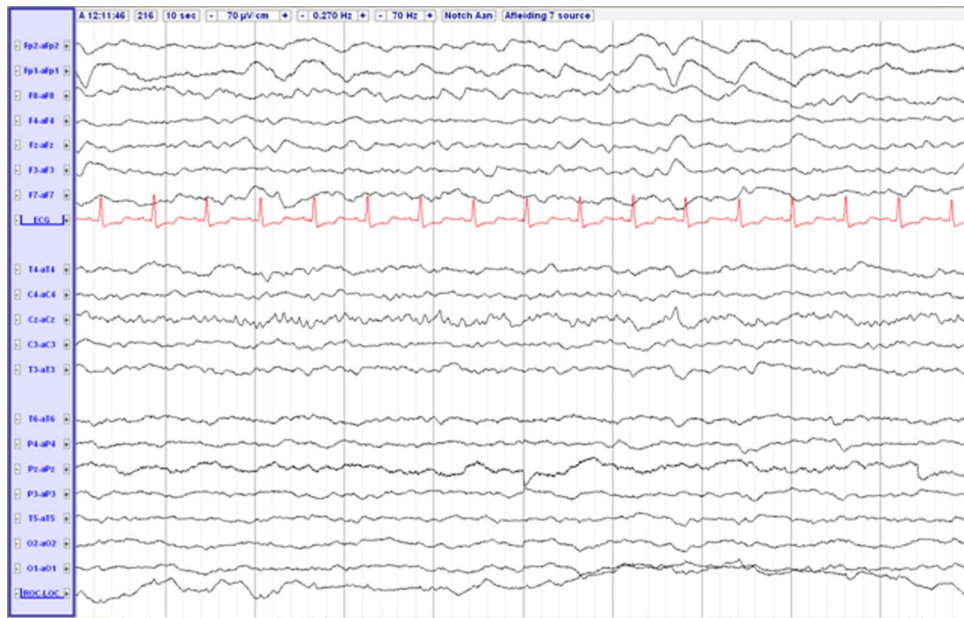


Figure 1 EEG on day of deterioration, 5 days after initial good recovery from anoxic insult, revealing sharp-wave complexes at the vertex.

Little is known about the true incidence of delayed postanoxic encephalopathy, ranging from a reported 1–28/1000 in patients experiencing a hypoxic event.^{2–5} A variety of symptoms may mark the unexpected neurological deterioration, which develops within 1–4 weeks; parkinsonism, tetraparesis, pseudobulbar palsy, sphincteric incontinence, apathy or agitation even up to manic behaviour.^{1–6}

The brain is susceptible to a variety of hypoxic causes; reduced global blood level of oxygen (drowning, airway obstruction, reduced inspired oxygen content), circulatory failure, carbon monoxide intoxication, hypoglycaemia (blocking oxidative metabolism and thereby halting intracellular respiration).⁷ How these events lead to delayed postanoxic encephalopathy, which is so typically marked by a lucid interval of ‘pseudorecovery’, remains to be discovered. Several pathophysiological mechanisms have been proposed throughout literature, none however conclusive. The reported findings at autopsy note extensive demyelination sparing the subcortical arcuate fibres, degeneration of striatum and caudate nucleus, no significant blood vessel abnormalities and accumulation of fat-laden macrophages.^{2–5} Neuroimaging also shows these typical lesions at basal ganglia and cortical laminar necrosis. CT may reveal a hypodense striatal region, whereas MRI is more sensitive and shows hyperintense signals in the basal ganglia or in the white matter in the arterial watershed zones and along the gyri, especially on fluid-attenuated inversion recovery and diffusion-weighted images.^{5 7–12} More advanced techniques such as

positron emission tomography-MRI and susceptibility-weighted MRI (SWI) look promising, but they are not available in most clinical settings.^{13 14}

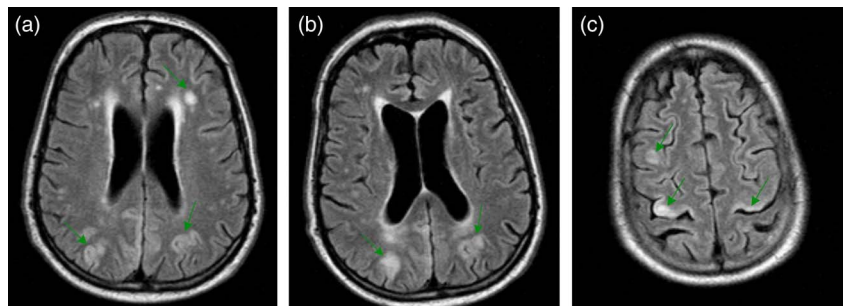
In the presented case the CT scan appeared normal, however subsequent MRI scans of the brain showed progressive cortical laminar necrosis.

Prognosis of delayed postanoxic encephalopathy is reportedly poor. The neurologic deterioration may progress to coma, death or severe disability, although some patients may have a second recovery period.^{2–5} Treatment is predominantly symptomatic.

Learning points

- ▶ Every patient who presents with changes in behaviour, motor responses or consciousness upon a lucid interval of seemingly good recovery from an anoxic insult, should be considered as possibly having a delayed postanoxic encephalopathy.
- ▶ Classically there is a lucid interval after initial good recovery from an anoxic insult, upon which neurological deterioration may progress to coma, death or severe disability.
- ▶ Other causes such as a non-convulsive status epilepticus must be ruled out and treated when diagnosed.

Figure 2 Fluid-attenuated inversion recovery-weighted MRI, revealing cortical laminar necrosis and periventricular white matter lesions, at 4 days after an anoxic insult (A) and 18 days on deterioration (B and C).



Competing interests None.

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