Postgraduate Medical Journal (1985) **61**, 253–254

Delayed encephalopathy following cardiac arrest

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Summary: A 46 year old woman suffered a post-operative cardiac arrest associated with prolonged depression of oxygenation and respiration. She made a good initial recovery but one year later insidiously developed symptoms of widespread central nervous system damage compatible with a delayed post-hypoxic encephalopathy. This case is unusual in the length of time between the hypoxic insult and the later deterioration and also illustrates other atypical features of a delayed post-hypoxic syndrome.

Introduction

The neurological features of hypoxic encephalopathy are usually evident shortly after the cerebral insult (Caronna, 1979). However, a number of reports have described an apparent recovery from hypoxia followed by deterioration at a later stage (Courville, 1957: Dooling & Richardson, 1976; Gordon, 1965; Plum *et al.*, 1962). In these cases the 'lucid' interval between the hypoxic episode and the onset of encephalopathy is usually days or at the most a few weeks. We report a patient who made a good recovery from an hypoxic episode secondary to a cardiac arrest and developed a progressive encephalopathy following an interval of greater than one year.

Case report

A 46 year old female assembly worker underwent a right hemicolectomy in January 1980 for persistent abdominal pain and obstructive episodes. Subsequent histological examination confirmed the diagnosis of Crohn's disease. Two days post-operatively she suffered a cardiac arrest related to disturbance of the serum potassium. It was with difficulty that the ventricular fibrillation was converted and respiration was still inadequate two hours later, requiring artificial ventilatory support for 24 h. Arterial blood gas analysis after conversion to sinus rhythm but before assisted ventilation gave a Po_2 8.82 kPa, Pco_2 5.54 kPa and pH 7.155. The systolic blood pressure one hour post-arrest was 60 mm Hg and two hours later a pressure of 104 mm Hg was recorded. After disconnection from artificial ventilation the patient was confused but proceeded to make a good recovery. Five days later she was examined by a neurologist and the only abnormal features were mild impairment of short term memory, slight dysarthria and a minor degree of gait ataxia. On examination six months later there had been sustained improvement and she had returned to work virtually asymptomatic.

In January 1981 she developed further dysarthria which became insidiously more pronounced. Intermittent painful spasms of the neck muscles followed and then similar spasms appeared in the muscles of the jaw and mouth, induced by eating and talking. These problems became disabling and she was reluctant to leave her house. Examination in April 1982 showed mild impairment of short term memory but no other disturbance of higher function. The verbal IQ was 92 and the performance IQ 96 which, on formal psychological testing, was felt to be compatible with her prior intellectual attainment. The gait was slightly shuffling. Eye movements were normal. The muscles of mastication were strong but activity of these muscles produced dystonic spasms of the jaw and mouth. The jaw jerk was pathologically brisk. The palate was paralysed although a gag reflex was present. Attempts at phonation produced marked dystonic spasms of the muscles of the jaw and the spastic dysarthric speech produced was virtually unintelligible. The neck muscles exhibited dystonic spasms both spontaneously and on clinical testing and the tongue appeared spastic. Cogwheel rigidity was evident at both wrists and a spastic increase of tone was found in both legs. The deep tendon reflexes were pathologically brisk but the plantar responses were

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Accepted: 28 March 1984

flexor. The glabellar tap sign was positive and pout, snout and palmarmental reflexes were all present. Rapid alternating movements and fine finger movements were impaired in both upper limbs in association with a degree of bilateral intention tremor on finger to nose testing.

Normal investigations included routine haematology, blood viscosity, biochemistry screening, copper and caeruloplasmin levels. The autoantibody screen and syphilis serology were negative. Both computed tomographic scan and electro-encephalogram showed no abnormality. The cerebrospinal fluid contained a marginally elevated protein of 0.5g/l but normal IgG/albumin ratio and no excess of cells. Visual evoked responses were also normal.

L-dopa with bensarizide was without effect on the dystonic disturbance and baclofen and diazepam were equally unhelpful. Haloperidol (7.5 mg/d) resulted in initial improvement but this has not been sustained and the dystonic spasms are an increasing problem.

Discussion

Previous reports of delayed encephalopathy have emphasized the importance of carbon monoxide induced hypoxia (Courville, 1957; Dooling & Richardson, 1976; Plum *et al.*, 1962) but in a few cases similar delayed deterioration has been observed following other hypoxic-ischaemic insults (Dooling & Richardson, 1976; Meriwether *et al.*, 1955; Plum *et al.*, 1962). A common factor in all these patients is the prolonged depression of oxygenation and respiration, as was evident following cardiac arrest in the present case. The striking feature in this patient is the duration of the interval between the hypoxic event and the onset of the encephalopathy. A comparable case in the literature describes a man of 48 y who developed signs of Parkinsonism 4 y after at least two episodes of unconsciousness related to occupational carbon monoxide exposure (Ringel & Klawans, 1972). In this case a precise connection between the Parkinsonism and the earlier carbon monoxide intoxication remains speculative.

The clinical features of the encephalopathy in the present case are also most unusual. Intellectual function has commonly been impaired in earlier reported cases, corticospinal tract disturbance is prominent and the cerebellum has appeared particularly susceptible to hypoxic damage (Caronna, 1979). Parkinsonian features are well described, particularly following carbon monoxide induced hypoxia (Ringel & Klawans, 1972). These neurological features have often arisen acutely, whereas in the present case the progression has been insidious and continues many months after the first symptoms were noticed. Furthermore, corticobulbar and extrapyramidal pathways appear to have been primarily affected, with the corticospinal tracts and cerebellum being involved to a lesser extent and with relative preservation of higher cerebral functions. The prominence of dystonia is remarkable, although an early description of delayed encephalopathy by Deutsch (1917) mentions dystonic posturing.

Assessment of this patient has failed to reveal any alternative pathological explanation for her present state and the authors feel justified in concluding that this is an unusual form of delayed post-anoxic encephalopathy. Others have speculated on the cause of this condition (Ginsberg, 1979), and vascular, demyelinating and toxic aetiologies have been suggested. Perhaps the most acceptable explanation involves a progressive demyelinating process initiated by the original hypoxic episode, but the clinical features here are atypical. Careful clinical and ultimately histological analysis of patients who have recovered from a cardiac arrest may further illuminate this intriguing entity.

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