CLINICAL REPORTS

Delayed Postanoxic Encephalopathy

C. A. HARDY, M.D.,* AND H. P. FISCHBACH, M.D.†

When neurologic examination immediately after an episode of cerebral hypoxia shows no abnormality, the prognosis is usually excellent. Rarely, a syndrome of delayed postanoxic encephalopathy may occur. The following report describes such a case following hypoxia during prolonged attempts to intubate the trachea.

REPORT OF A CASE

A healthy 20-year-old man had a lacerated right lingual artery following dental surgery. Marked edema, bleeding and distortion of the glottis were observed during direct laryngoscopy. The patient refused elective tracheostomy despite repeated unsuccessful attempts at awake nasotracheal intubation.

Subsequently, with the patient breathing spontaneously, a rapid induction sequence with 40 mg thiamylal and 100 mg succinylcholine was performed. Intubation was unsuccessful with an endotracheal tube but was subsequently accomplished with a rigid bronchoscope. Cyanosis was present for 4-6 minutes prior to intubation. Systolic blood pressure was more than 100 torr immediately before and after attempted intubation, and pulse rate was 48/min. While \( O_2 \) was given via the bronchoscope, a tracheostomy was performed. Then halothane, \( N_2O \) (60 per cent), and \( O_2 \) were administered for a right neck exploration, ligation of the right lingual artery, and repair of a bronchoscopic laceration of the right tonsillar pillar. Operating time was 3½ hours, during which ventilation was assisted.

Postoperatively, the patient responded normally to verbal stimuli, and the pupils were equal and responsive to light. Because of persistent bleeding, 4 hours later the patient again underwent anesthesia and operation which were uneventful. Three hours after this procedure, the patient was "fully responsive, alert and cooperative."

Later that day, he had spastic movements of the right hand, followed by a generalized seizure, with urinary incontinence but no tonic-clonic movements. Neurologic examination revealed generalized severe weakness and tremors of all extremities, without unconsciousness. Sensation was intact and funduscopic examination disclosed no abnormality. The next day, the patient lost movement of his right hand, but had good tone in all other muscles. Right upper extremity reflexes were greater than those on the left. Bilateral extensor plantar reflexes, loss of speech, and decerebrate posturing were noted. Funduscopic examination again disclosed no abnormality. Cerebrospinal fluid was also normal. During the next three days, lethargy progressed to stupor. Three days later, vertebro and carotid angiography disclosed no abnormality.

Ten days postoperatively, the patient had full quadriplegia, with no purposeful responses and intense chewing motion. Five days later, spontaneous movement of the extremities reappeared, and the patient's condition progressively improved. Follow-up evaluation six months post-operatively showed intellectual ability (as evidenced by I.Q. scores) not significantly diminished from pre-induction military test scores. The patient's mental status is normal, but spastic quadripareis and dysthria persist.

DISCUSSION

In 1962, Plum and Posner⁴ reported 14 cases of delayed postanoxic encephalopathy. The syndrome was first related to carbon monoxide poisoning, but has also been described to occur following anesthesia, cardiac arrest, hypoglycemia, and heroin overdose. Following an anoxic insult, a lucid interval of 2–21 days occurs, during which the patient appears to have recovered completely. No sign of neurologic deficit is present during the lucid interval, but onset of the encephalopathy is marked by "irritability," apathy, and confusion. Because of the intervening period of normoxia, a diagnosis of hysterical reaction is sometimes considered. Development of shuffling gait, clumsiness, and diffuse skeletal muscle spasticity or rigidity resembling parkinsonism precede coma and death. The EEG commonly shows non-focal abnormalities.

Postmortem examination of the brains of

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* Instructor, Department of Anesthesia, University of Pennsylvania, School of Medicine, Philadelphia, Pennsylvania 19104.
† Resident, Navy Regional Medical Center, Philadelphia, Pennsylvania.

Accepted for publication June 29, 1975.

Address reprint requests to Dr. Hardy.
patients who have had this syndrome shows diffuse, marked demyelination involving the cerebral hemispheric white matter, often extending across the corpus callosum. Infarction of the basal ganglia is sometimes seen. However, pathologic changes are commonly found in sections of the basal ganglia of persons who have undergone anoxic episodes without clinically recognized neurologic sequelae. Speculation as to the specific mechanism causing demyelination includes allergic encephalitides, cerebral vasculitis, cerebral edema, and poisoning of an enzyme system involved in the production of myelin. Although observed in primates experimentally subjected to carbon monoxide in hypoxic concentrations, its occurrence is not consistent.

Our patient had typical postanoxic encephalopathy except for the focal onset of his symptoms. It can be postulated that the focal signs were caused by injury of the left carotid artery by the bronchoscope, resulting in either spasm or clot formation and embolization. While this might have occurred during the onset of the patient's symptoms, the clinical impression formulated during his subsequent course remained that of postanoxic encephalopathy.

Neither prevention nor treatment of the syndrome has been established, but Plum et al. noted a possible correlation of the syndrome with early ambulation. They, therefore, treated 100 severely anoxic patients with ten days of strict bed-rest, without occurrence of the syndrome. However, their own critique states that the incidence of delayed postanoxic encephalopathy is less than 1 per 1,000 cases of anoxia.

Until more is learned about this syndrome, symptomatic treatment seems most logical. During the diffuse inflammatory state, cerebral edema should be demonstrated or ruled out by appropriate diagnostic studies. When increased pressure is present, treatment with mannitol, steroids, and hypothermia is in order if cerebral edema is present. After development of the syndrome, sedation and bed-rest until neurologic damage has been resolved are also suggested.

The authors thank Dr. Harvey M. Shapiro for help in the preparation of this manuscript.

REFERENCES