

Cortical Blindness After Electroconvulsive Therapy

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Electroconvulsive treatment (ECT) for mental disorders is considered to be safe and effective when administered according to standard protocols for appropriate indications.¹⁻⁴ It can produce transient cognitive impairment, but not structural damage, to the brain and is remarkably free from serious complications. Deaths after ECT are reported to occur at a rate of 2 per 100,000 treatments, about the same as for general anesthesia,⁵ and a task force of the American Psychiatric Association stated that there are no absolute contraindications to ECT.⁶

Bilateral occipital lobe brain hemorrhages also occur rarely; we found only six cases reported during the last two decades. None were caused by seizures, hypertension, trauma, or ECT.⁷⁻¹²

We report here a case of a man who had bilateral occipital lobe hemorrhages for which he was treated neurosurgically 9 days after receiving two electroconvulsive treatments for a severe depressive disorder that had been unresponsive to drug treatment. Based on the results of searches of MEDLINE and HEALTH databases, as well as on a review by Devanand et al⁴ of ECT complications occurring before the existence of these databases, we believe this report to be the first of its kind.

Case Report

Our patient, a 46-year-old man, was admitted to the hospital with complaints of severe, generalized headache and loss of vision in both eyes. His headache began shortly after he received a second ECT 9 days earlier, and his vision diminished 2 days before admission. On the day of admission, his vision loss was total.

He was known to be an intermittent intravenous heroin user since the age of 16 years, but he had stopped 2 years before his admission and

was participating in a methadone maintenance program, taking 70 mg of methadone daily. He also had a history of cocaine sniffing six or seven times in the last 10 years, but he reported no cocaine use during the 8 months before receiving ECT.

He had become clinically depressed 4 years earlier, after the death of a 16-year-old son, and was prescribed paroxetine and alprazolam. His condition had worsened recently, and ECT was recommended by his physicians. ECT was administered in a medical facility staffed by those experienced with the procedure, according to a standard protocol using sedation, anesthesia, succinylcholine, oxygen, and appropriate monitoring. An adequate grand mal seizure was achieved lasting 30 to 40 seconds. His medical history revealed no evidence of hypertension, diabetes mellitus, or heart disease. A test for human immunodeficiency virus infection was negative 1 year earlier.

The patient was working as a computer programmer and was married to a caring and medically cooperative spouse. His paternal family history was unavailable, but his maternal relatives were known for longevity, and there was no history of chronic medical diseases.

When examined in the emergency department he was alert and fully oriented. His weight was 180 pounds, height 70 inches, blood pressure 133/85 mmHg, pulse rate initially 76 beats per minute, which later slowed to 47 beats per minute, respirations 16/min, and temperature 98.1°F.

He was unable to perceive light or recognize large objects and did not react to visual threats. Both pupils reacted to light, and corneal reflexes were normal. Eye movements were normal to commands, and there was no deviation of gaze. The physical appearance of the eyes was normal. An ophthalmologist confirmed these findings and performed dilated funduscopy, which revealed normal optic discs, retina, and media. There was no evidence of trauma to the head, skin rashes, or new needle tracks. His neck was supple and without jugular venous distention, lymphadenopathy, or thyromegaly. His lungs were normal, and his

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heart was normal in size, rhythm, and sounds. The abdomen showed no tenderness, masses, or organomegaly, and peripheral pulses were normal.

His speech and gait were normal and cranial nerves intact, except N-II. There was no sensory or motor loss in his extremities, and his deep tendon reflexes were symmetrically reactive.

There were normal findings on complete blood count, and his platelet count was normal, as were the results of coagulation tests and a urinalysis. His metabolic profile was normal except for slightly elevated aspartate aminotransferase (44 U/L) and alanine aminotransferase (53 U/L) levels. A hepatitis profile was positive for hepatitis B core antigen. An electrocardiogram showed only sinus bradycardia (47 beats per minute), and findings on chest radiography were normal.

Computerized tomography of his head, without contrast, showed intracerebral hemorrhages in both occipital lobes without other abnormal findings. Findings on Doppler sonography of the carotid arteries were normal.

A diagnosis of cortical blindness from bilateral occipital hemorrhages was made, and the patient was admitted to an intensive care unit and taken to surgery for evacuation of the occipital clots, which was performed without complications. His vision began to improve within 24 hours, but on the 3rd postoperative day he developed signs of alprazolam withdrawal and was prescribed alprazolam and haloperidol. Methadone maintenance was continued. Holter monitoring of heart rate and rhythm for 24 hours showed sinus rhythm, normal conduction with occasional premature atrial contractions, and one 5-beat run of ventricular tachycardia.

He recovered from surgery and was discharged home on the 8th postoperative day. His vision improved steadily and was 20/20 bilaterally by the 15th day. He had visual scotomata and dyschromatopsia for a few weeks, but these eventually disappeared. He also had agraphia, which resolved by the 12th day. Six months later he felt fine and his vision was normal.

Discussion

The overall safety of ECT is supported by many studies, which were reviewed by Devanand et al in 1994.⁴ Even before the adoption of modern methods of ECT administration, autopsy studies from the 1940s and 1950s failed to show struc-

tural changes in the brain that could not be explained by agonal changes or concomitant diseases. Intracranial aneurysms are not an absolute contraindication when appropriate monitoring and control of blood pressure are available.¹³ There is a case report of prolonged paralysis after ECT in a patient who had attempted suicide by ingesting an organophosphate compound.¹⁴ Also, there was a report of common migraine headaches precipitated by ECT, perhaps in the same manner as spontaneous seizures.¹⁵ Nevertheless we were unable to find any report of intracranial bleeding after ECT.

Intracerebral bleeding is usually caused by hypertension, cerebral amyloid angiopathy, bleeding-into-brain tumors, coagulopathies (especially from the use of anticoagulants or thrombolytic agents), rupture of vascular malformations, vasculitis, and the use of sympathomimetic drugs (amphetamines, cocaine, and phenylpropanolamine).^{16,17} Bleeding into the occipital lobes is uncommon, however. We found only six cases of bilateral occipital bleeding during the last 20 years,⁷⁻¹² mainly from unusual causes, such as those related to pulmonary sarcoidosis⁸; bacterial endocarditis with rupture of bilateral mycotic aneurysms in the occipital lobes⁹; a combination of Alzheimer dementia, amyloid angiopathy, and an old unilateral parieto-occipital hemorrhage¹⁰; and eclampsia.⁷ The cause of old hemorrhages in a case of an 80-year-old patient was not clear.¹¹ Interestingly enough, Iob et al¹² observed that hemorrhages were caused by accidental high-voltage (1000 V) electrocution applied to the occipital area (direct thermal and mechanical impacts were excluded).

Because our patient was a known narcotic user for 30 years, on methadone maintenance for 2 years, and admitted to sniffing cocaine a few times, we interviewed him and his spouse repeatedly for details about his drug use, but we discovered no additional information that implicated sympathomimetic drugs as a possible cause of his occipital hemorrhages. Moreover, as measured by his physical and laboratory examinations, he had no other predisposing conditions and was in good health except for his depressive disorder.

We do not claim that this case proves a cause-and-effect relation between ECT and bilateral occipital hemorrhages, but it presents an extraordinary convergence of events, and the possibility of a causal relation must be considered.

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