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Delayed Cortical Blindness after Electroconvulsive Therapy in a Healthy Young Patient: A Case Report

*Han Wu[1,2], Li-na Jia[1,2], Wei Jiang[1,2], Yi-lang Tang[3,4], Yan-ping Ren[1,2] and Xin Ma[1,2]**

[1]The National Clinical Research Center for Mental Disorders & Beijing Key Laboratory of Mental Disorders, Beijing Anding Hospital, Capital Medical University, China

[2]Advanced Innovation Center for Human Brain Protection, Capital Medical University, China

[3]Department of Psychiatry and Behavioral Sciences, Emory University School of Medicine, USA

[4]Mental Health Service Line, Atlanta VA Medical Center, USA

Abstract

We report a 19-year-old healthy young patient diagnosed with schizophrenia who developed cortical blindness three hours after the initial ECT. After extensive workup, patient was diagnosed with pneumonia with altered mental status, along with blindness. The alveolar-arterial oxygen partial pressure (PA-aDO$_2$) increased significantly. Supplemental oxygen was given. Mannitol was administered for dehydration; cefuroxime and dexamethasone were given intravenously. His vision fully recovered 15 h after ECT. The interaction between respiratory infection and ECT might contribute to local vasospasm of occipital lobe, leading to cortical blindness. For patients with respiratory tract infection after ECT, especially with significantly increased PA-aDO$_2$, antibiotic treatment and dehydration should be administered to prevent the occurrence of cortical blindness.

Keywords: Electroconvulsive therapy; Delayed cortical blindness; PA-ADO$_2$

Introduction

Electroconvulsive Therapy (ECT) is an important somatic treatment that stimulates the brain with a short and appropriate amount of electric current to induce generalized convulsions. Anesthetic and muscle relaxant are administered to increase the safety [1,2]. It is a highly efficacious treatment for certain psychiatric disorders including major depression, bipolar disorder and schizophrenia [3,4]. The common side effects and complications related to ECT include cognitive and cardiovascular side effects, post-ECT confusion [5,6]. Rare complications such as cortical blindness after ECT have been reported [7-10]. Most reported cases of transient blindness occurred immediately after ECT. Here we report a case of delayed cortical blindness that occurred three hours after the initial ECT.

Case Presentation

A 19-year-old male with no known prior medical illnesses was hospitalized due to psychotic symptoms and his admission diagnosis was schizophrenia. Admission laboratory test results were all within normal limits except prolactin (35.0 ng/ml, normal range: 2.5 ng/ml to 17.0 ng/ml). His head CT showed slightly widened choroid fissures and cerebellar creek-cerebellar medulla oblongata. The hippocampal volume was small. Head MRI on admission showed no abnormalities. Trihexyphenidyl (4 mg bid) was used to improve the extra pyramidal side effects caused by risperidone taken prior to admission. Olanzapine (15 mg bid) was used to treat psychotic symptoms but with limited improvement and ECT treatment was deemed appropriate. Before ECT, the Complete Blood Count (CBC) with differential, electrocardiogram and chest X-ray examination were all unremarkable. Routine admission physical examination was also unremarkable. The patient was administered the following medications intravenously: Atropine (0.25 mg) 30 min before ECT, propofol (70 mg) for anesthesia, and succinylcholine (24 mg) for muscle relaxation. Bitemporal electrodes were placed and a brief-pulse constant current apparatus (Somatics Thymatron®, Venice, FL, USA) was used [11,12]. The stimulus settings at initial ECT were 900-mA pulse amplitude, 1-millisecond pulse width, 30-Hz pulse frequency, and 1.4-second stimulus duration; electrical energy setting at 15 joules (electrical energy setting method: Age-based method at 80% of the age); the delivered charge was 74 mC and the motor seizure duration was 130 sec. The patient was oxygenated with pure oxygen during the procedure and the blood oxygen saturation was 99%. The patient regained

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Correspondence:
Xin Ma, The National Clinical Research Center for Mental Disorders & Beijing Key Laboratory of Mental Disorders, Beijing Anding Hospital, Capital Medical University, 5 Ankang Lane, Deshengmenwai Avenue, Xicheng District, Beijing, 100088, China, E-mail: maxinanding@ccmu.edu.cn

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consciousness approximately 12 min after ECT. He walked back to the inpatient unit accompanied by a nurse and had lunch in the unit. Approximately 3 h after ECT (1 h after lunch), the patient suddenly became anxious and panicked. He complained of a sudden bilateral vision loss, headache, nausea, and he vomited once without ejection. His temperature was 38.7°C (101.3 F). He became confused gradually. Physical examination showed corneal reflex disappeared while pupils were normal size (2.5 mm) and the pupillary light reflex was normal. Neurological examination showed Hoffmann signs were positive bilaterally and no other abnormal signs were found. Head MRI showed no abnormality. Headaches and nausea persisted and he was transferred to the emergency department 4 hours after the onset of blindness. He head CT showed no obvious abnormalities. He became more and more confused. Ophthalmic examination found that the optic disc was round with a clear boundary; the retina had no hemorrhage, exudation, or edema. Retinal blood vessels were normal. The light reflex was present in the central fossa of macula. The chest X-ray suggested bilateral pulmonary infection. Laboratory results showed an increase in white blood cells 16.06 × 10^9/L) and the percentage of neutrophils (Neutrophils 13.44 × 10^9/L, neutrophil % 83.7%) and decrease in lymphocytes (7.92%). Arterial blood gas analysis (12 h after the onset of blindness) showed a significant increase of alveolar-arterial oxygen partial pressure (PA-aDO_2, 64.2 mmHg) (normal range: 5 mmHg to 15 mmHg). He was diagnosed with pneumonia with altered mental status by the ER physician. Supplemental oxygen was given. Mannitol (250 ml) was administered for dehydration, and cefuroxime (1.5 g) and dexamethasone (10 mg) were given intravenously. Approximately 15 h after ECT, the patient reported his vision had recovered. Repeat complete blood counts and blood gas analysis were within normal ranges and he was transferred to the unit. The treatment team decided to discontinue ECT and continue olanzapine (15 mg/d) treatment. His symptoms showed mild improvement and he was discharged 1 month later.

Discussion

The important feature of this case report is that transient blindness occurred 3 h after ECT in a healthy young patient with no pre-existing medical conditions. His vision recovered 15 h after the onset of blindness through treatment. Prior to ECT, he was on olanzapine (30 mg/d) and trihexyphenidyl (8 mg/d). He had never received ECT before. ECT treatment parameters were bitemporal electrode placement, 74 mC electric charge, 15 joules energy, and the induced seizure time was 130 sec. His corneal reflex disappeared and pupil light reflex was intact; WBC and NEUT% increased and LYM% deceased; PA-aDO_2 increased significantly. Oxygenation, mannitol, steroid, and cefuroxime were effective for the blindness. There have been three cases of cortical blindness after ECT reported previously [8-11]. Our case, along with the three previous reports, is consistent with cortical blindness associated with ECT, which has the following features: (1) It usually occurred post the initial ECT treatment; (2) the seizure duration was often long, mostly longer than 120 sec; (3) the typical symptoms include the disappearance of the corneal reflex, but the light reflex was intact; (4) No abnormality was found in head CT, MRI and ophthalmic examination; (5) Blindness generally resolves within 24 h after ECT. The manifestation that the pupil light reflex was intact while the corneal reflex disappeared, and that no optic nerve edema and papillary edema found, plus negative findings on brain imaging, all suggest that the optic nerve-midbrain oculomotor nerve circuit was intact. There are several possible mechanisms that may explain post-ECT blindness. First, it may be associated with idiopathic or secondary focal or generalized epileptic seizures [11]. The blindness may be an ictal rather than a postictal phenomenon, symptomatic of non convulsive status epilepticus [3]. The patient regained consciousness after ECT and recalled the events happened after ECT. In our case, although he did not undergo EEG examination after blindness, his cortical blindness caused by non-convulsive status epilepticus was unlikely. Second, it may be caused by cerebral organic diseases, such as cerebral infarction, cerebral hemorrhage, and Transient Ischemic Attack (TIA) [13]. However, no abnormality was found on the head CT or MRI in this case. The patient was a healthy young male with no high-risk factors for cerebrovascular disorders. Additionally, transient blindness may also be caused by psychological factors, such as separation and conversion disorder [15]. However, most cases with transient blindness associated with conversion disorder retain their mobility and perform well in motor performance. But our patients had no prior history to suggest any risk factors, and his physical examination showed that the blink reflex disappeared. Among the possible mechanisms, we think cerebral vasospasm is the likely explanation for the case in the report. Cerebral vasospasm caused by ECT was related to electrical stimulation during treatment. In this case, the stimulation energy was high and the seizure time was long, which was a risk factor for cerebral vasospasm [12,13]. Symptoms such as headache, nausea and vomiting often occur immediately after ECT. Different from previous reports, the blindness in this case occurred 3 h after ECT. It suggested that the cortical blindness in this patient may not be directly caused by ECT-induced vasospasm. We speculate that the combined action of respiratory infection and pulmonary oxygen exchange dysfunction may have contributed to vasospasm. The respiratory infection in this is consistent with aspiration pneumonia. A previous case report had also confirmed that respiratory infection can cause cortical blindness [16,17]. The interaction between respiratory infection and ECT might contribute to local vasospasm in the occipital lobe, leading to delayed-onset blindness in this case.

In conclusion, although rare, cortical blindness may happen after ECT. The high energy of ECT simulation and respiratory infection may have contributed to it. To minimize the risk, we suggest using lower energy settings, or use the stimulus titration method. If blindness occurs, searching for and treating medical conditions (in this case pneumonia), plus treatments targeting brain edema may be effective. For patients with respiratory tract infection after ECT, especially with significantly increased PA-aDO_2, antibiotic treatment and dehydration should be administered to prevent the occurrence of cortical blindness.

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