Interdisciplinary Neurosurgery: Advanced Techniques and Case Management 9 (2017) 89–91

Contents lists available at ScienceDirect

Interdisciplinary Neurosurgery: Advanced Techniques and Case Management

journal homepage: www.inat-journal.com

Case Report & Case Series

Intraparenchymal hemorrhage after electroconvulsive therapy☆

Elizabeth W.J. Carlson [a], Howard Weeks [b], William T. Couldwell [a], M. Yashar S. Kalani [a,*]

[a] Department of Neurosurgery, University of Utah School of Medicine, Salt Lake City, UT, United States
[b] Department of Psychiatry, University of Utah School of Medicine, Salt Lake City, UT, United States

ARTICLE INFO

Article history:
Received 9 June 2017
Revised 10 July 2017
Accepted 16 July 2017

Keywords:
Electroconvulsive therapy
Tonic-clonic seizure
Intracerebral hemorrhage

ABSTRACT

A 46-year-old woman with a history of medically refractory depression experienced a tonic-clonic seizure shortly after electroconvulsive therapy for severe depression. The patient was stabilized, diagnostic imaging was performed to exclude an underlying vascular lesion responsible for the hemorrhage, and the patient underwent decompressive hemicraniectomy and evacuation of hematoma. Despite aggressive measures, however, the patient ultimately died of cardiopulmonary complications of the inciting hemorrhage. This report highlights a rare but potentially devastating complication of electroconvulsive therapy.

1. Introduction

Electroconvulsive therapy (ECT) is a safe and well-tolerated treatment paradigm for patients with medically refractory depression. In one large series involving 20,000 ECT procedures administered over a 3-year period, there were four fatalities [3]. These included a case of coronary occlusion, one case of aspiration pneumonia, a case of pulmonary embolism, and one case of intracerebral hemorrhage, which was attributed to venous occlusion. Other well-documented complications of ECT include intra-abdominal hemorrhage caused by mesenteric injury [2] and ruptured viscus. Intracranial hemorrhage is a rarely reported complication of ECT, but is generally subdural in nature, likely caused by rupture of bridging veins during administration of shock [4]. A case of ischemic stroke after administration of ECT has been reported [1]. Patients undergoing ECT are not routinely subjected to imaging of their brain to rule out underlying structural defects or intracranial pathology such as tumor. We present a case of a fatal intracerebral hemorrhage in a patient after ECT for severe depression.

2. Case report

A 46-year-old woman with a history of medically refractory depression (Quick Inventory of Depressive Symptomatology Self-Report score 26; consistent with severe depression) was evaluated by our psychiatry service for ECT treatment. The patient's family history was significant for depression, anxiety, and schizophrenia. The patient had no significant social history. Her medical history was notable for asthma, depression, diabetes mellitus, and chronic headaches. The patient's medications included bupropion XL (300 mg daily), clonazepam (2 mg three times per day), fluvoxamine (200 mg daily), lamotrigine (100 mg daily), propranolol (20 mg daily), and quetiapine (50 mg daily).

The patient's pre-ECT examination documented decrease in affect, mood, anxiety, voice inflection, rate of speech and speech content, and psychomotor activity. The patient underwent 2 rounds of ECT stimulations. Stimulation 1 consisted of bifrontal stimulation for 6 s at 90 Hz, 0.8 amps, 0.37 msec. Hyperventilation was used. The charge was 319 mC; energy: 69.8 J; static impedance: 530 Ω, dynamic impedance: 267 Ω. The patient had a peripheral seizure at 21 s and central seizures at 27 s; postictal suppression was present. Stimulation 2 was augmented with 125 mg intravenous caffeine and hyperventilation. Again bifrontal stimulation was used for 7 s at 100 Hz, 0.8 amps, 0.37 msec. The charge was 414 mC; energy: 77.6 J; static impedance: 480 Ω, dynamic impedance: 236 Ω. There was a peripheral seizure at 71 s and central seizures at 128 s; postictal suppression was immediate.

During recovery in the postanesthesia care unit, the patient became agitated and received 5 mg midazolam, 2 mg lorazepam, and 5 mg haloperidol. She had a tonic-clonic seizure lasting 10 min. An immediate computed tomography (CT) scan revealed a large left basal ganglia intraparenchymal hemorrhage with intraventricular extension (Fig. 1A).

Upon identification of hemorrhage, the neurosurgery service was consulted. The patient was intubated and transferred to the intensive care unit, where she was noted to be localizing bilaterally but weaker on the right side. The patient's pupils were equal, round, and reactive to light. During the preparation for vascular imaging, the patient suddenly experienced pulseless electrical activity. After 30 min of

☆ Financial support: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

* Corresponding author at: Department of Neurosurgery, University of Utah School of Medicine, 175 North Medical Drive East, Fifth Floor, Salt Lake City, UT 84132, United States.
E-mail address: Neuropub@hsc.utah.edu (M.Y.S. Kalani).

http://dx.doi.org/10.1016/j.inat.2017.07.012

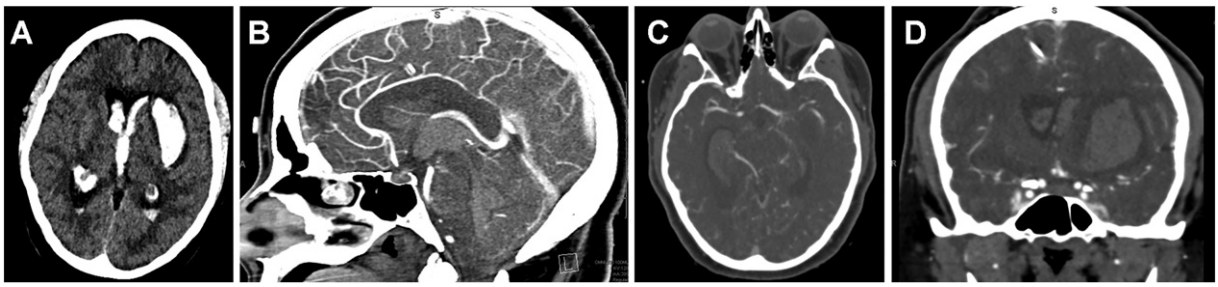

Fig. 1. (A) Axial noncontrast computed tomography demonstrates a large left basal ganglia intraparenchymal hemorrhage with intraventricular extension. (B) Sagittal, (C) axial, and, (D) coronal computed tomographic angiography demonstrates normal vasculature without evidence of aneurysms or vascular malformations.

resuscitation, a normal rhythm resumed. An external ventricular drain was placed, and intracranial pressures were noted to be elevated over 60 mmHg. The patient's postresuscitation examination was consistent with the admission examination. En route to the CT scanner, the patient developed pupillary asymmetry, which responded to mannitol. Repeat CT demonstrated interval increase in the size of the intraparenchymal hemorrhage (not shown). Computed tomographic angiography did not demonstrate a structural vascular cause responsible for the patient's hemorrhage (Fig. 1B–D).

The patient was taken for an emergent hemicraniectomy and evacuation of the hematoma. The procedure was uneventful, but during the closure of the skin, the patient experienced recurrent pulseless electrical activity, requiring over 30 min of resuscitation. Again, normal rhythm was restored. The patient's postoperative examination was consistent with brisk localization on the left side and brisk withdrawal on the right side. A formal diagnostic cerebral angiogram did not demonstrate any underlying vascular cause responsible for the patient's hemorrhage (Fig. 2). Similarly, a magnetic resonance imaging study did not reveal tumor or other causes responsible for this hemorrhage (Fig. 3). Despite aggressive medical therapy, the patient developed severe pulmonary edema that was recalcitrant to therapeutic interventions. Ultimately, after family discussion, the decision was made to withdraw care. The patient died 12 days after the original hemorrhage.

3. Discussion

Intracerebral hemorrhage after ECT is an exceedingly rare complication. In a series of 20,000 ECT procedures, there was a single case of intracerebral hemorrhage, meaning this complication is expected to occur in 0.005% of cases [3]. Indeed, there is only a single report of intracerebral hemorrhage after ECT [6] and a handful of cases of subdural hemorrhages in the literature [4]. These numbers support the belief that ECT is generally safe and well tolerated. As such, many practitioners do not routinely obtain intracranial imaging prior to administering ECT to patients. The patient in this report did not have prior intracranial imaging. Post-ECT imaging, which included CT angiography, MRI, and formal angiography, did not reveal an underlying cause for hemorrhage in our patient, meaning that even if the patient had had an imaging workup, it would have been impossible to predict the devastating outcome encountered.

A large study of incidental findings on MRI brains in the general population, which involved 2000 subjects, noted an incidental cerebral

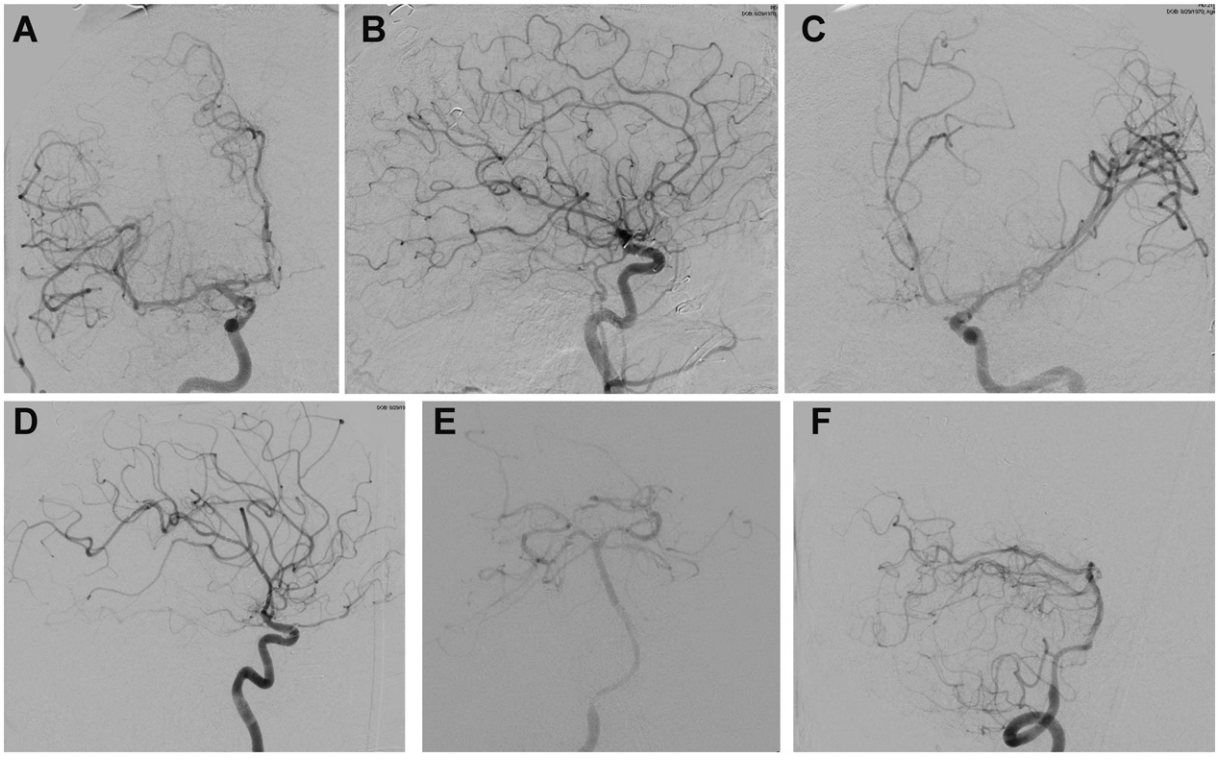

Fig. 2. (A) Anteroposterior and (B) lateral views of the right internal carotid artery; (C) anteroposterior and (D) lateral views of the left internal carotid artery; and (E) transfacial and (F) lateral views of the right vertebral artery demonstrate normal intracranial vasculature without evidence of abnormalities responsible for this patient's hemorrhage.