

# Acute Embolic Stroke After Electroconvulsive Therapy

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**Abstract:** This is the case report of a 44-year-old woman presented with an acute stroke immediately after electroconvulsive therapy (ECT). The patient had no significant medical history other than chronic depression. She was taking sertraline, and she had had multiple previous ECT treatments without any complications. While being monitored in the recovery room within 10 minutes after the last ECT session, she was found to have sudden onset of left-sided flaccid hemiplegia and numbness along with slurred speech. On arrival to our hospital, she was found to have flaccid hemiplegia on the left side involving the face, arm, and leg (face and arm more than the leg involvement), severe dysarthria, and mild neglect syndrome (National Health Institute Stroke Scale of 14). Noncontrast computed tomography (CT) of the head showed no signs of early ischemia, and iodine contrast CT angiography revealed right middle cerebral artery (MCA) (distal M1 segment) clot. Patient received intravenous recombinant tissue plasminogen (rt-PA) at 2.5 hours after the onset of symptoms, and then a total of 3.0 mg of intra-arterial (IA) rt-PA. Angiography at the end of the procedure showed successful recanalization of the M1 segment and normal vessel caliber with adequate distal flow. After the procedure, the patient made rapid improvements in all of her initial symptoms during the first 24 hours. An extensive stroke workup failed to reveal any cause of the stroke, including usual stroke and hypercoagulable risk factors. This was an acute embolic stroke immediately following an ECT, and without the aggressive thrombolytic therapy, the patient's outcome would have been poor because there was an M1 segment clot with a major MCA syndrome with relatively high National Institute of Health Stroke Scale. The neurological side effect profile of ECT is reported to be minimal with most common symptoms being headache, disorientation, and memory complaints. There is no clear cause-and-effect relationship in this case, and the stroke after ECT is extremely rare. In such rare event of stroke while receiving ECT, there is an effective treatment available using both intravenous and IA thrombolysis as reported in this case.

**Key Words:** acute stroke, cryptogenic stroke, electroconvulsive therapy

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A 44-year-old woman presented with an acute stroke immediately after ECT. The patient had no significant medical history other than chronic depression. This includes all the major risk factors for stroke such as hypertension,

hypercholesterolemia, and history or family history of stroke, as well as other rare hypercoagulable state workup. She was taking sertraline, and she had had multiple previous ECT treatments without any complications. While being monitored in the recovery room within 10 minutes after the last session, she was noted to have sudden onset of left-sided flaccid hemiplegia and numbness along with slurred speech. On arrival to our hospital, she was found to have flaccid hemiplegia on the left side involving the face, arm, and leg (face and arm more than the leg involvement), severe dysarthria, and mild neglect syndrome (National Health Institute Stroke Scale of 14). Noncontrast CT of the head showed no signs of early ischemia, and iodine contrast CT angiography revealed right MCA (distal M1 segment) clot (Figs. 1 and 2).

The patient received rt-PA intravenous infusion beginning at 2.5 hours after the onset of symptoms. There was no immediate clinical improvement, and she was taken to interventional suite for IA intervention.

A total of 3.0 mg of IA rt-PA was injected around the proximal and distal borders of the clot. Mechanical disruption of the clot was achieved using an agility-14 microguidewire. Angiography at the end of the procedure showed successful recanalization of the M1 segment (4B) and normal vessel caliber with good distal flow (Fig. 3).

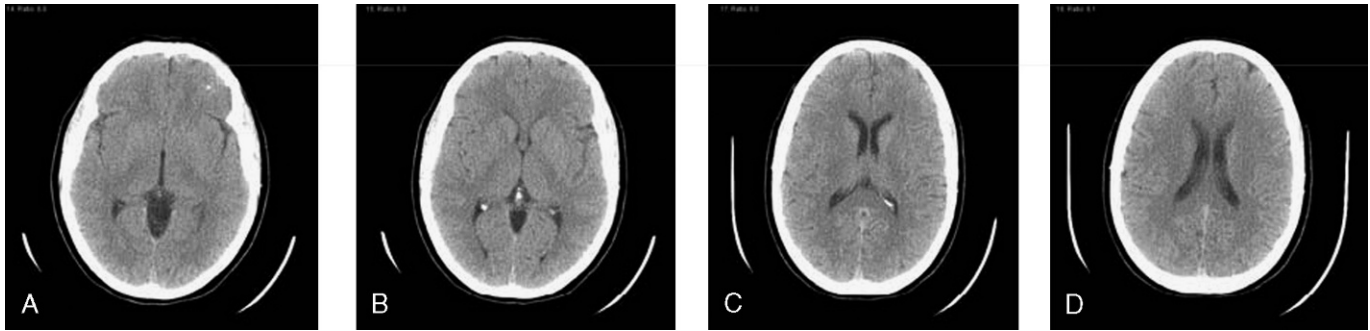
The patient tolerated the procedure well, and there were no procedure-related complications. While being monitored in the neurological intensive care unit, she made rapid improvements in all of her initial symptoms during the first 24 hours. At discharge to a rehabilitation hospital, she was only minimally symptomatic due to mild hemiparesis on the left side with no other deficit.

An extensive evaluation for etiologies underlying her stroke including echocardiogram of the heart, tests of hypercoagulability, and images of the head and neck failed to reveal any evidence of atherosclerosis or other causes for her stroke. The acute M1 occlusion and otherwise negative evaluation were felt to be most consistent with cryptogenic embolism.

## DISCUSSION

The patient had an acute embolic stroke immediately following an ECT procedure. Without the aggressive thrombolytic therapy, her outcome would have been poor because there was an M1 segment clot with a major MCA syndrome with relatively high National Institute of Health

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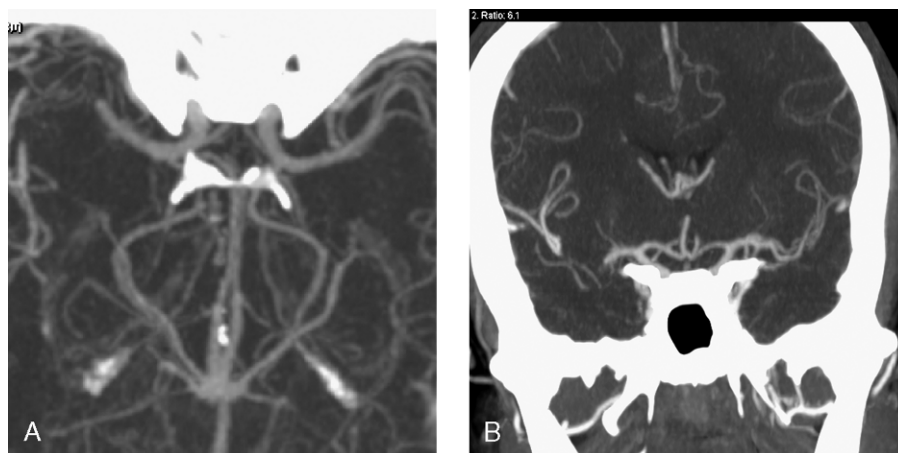


**FIGURE 1.** Initial axial CT images of the brain showing no signs of early ischemic changes before the administration of intravenous rt-PA.

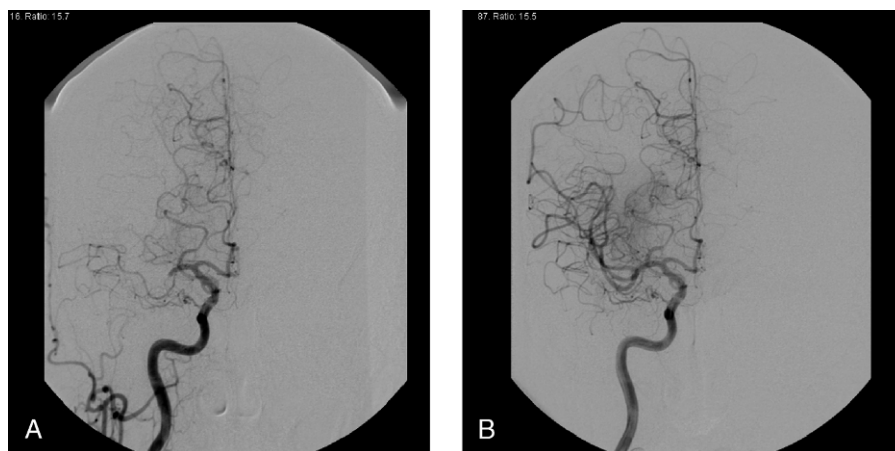
Stroke Scale. In the ECT literature, a case of reversible ischemic neurological deficit has been reported which was observed after ECT.<sup>1</sup> People who have known preexisting atrial fibrillation (AF) are commonly treated with long-term anticoagulation to reduce the risk of embolic stroke. Anticoagulation is strongly recommended for patients who are not being anticoagulated before ECT since there are documented reports of cardioversion during ECT.<sup>2,3,4</sup> There is no report in the literature whether ECT can directly cause an embolic stroke in patients with AF, however. Other potential effect of ECT is alteration in cerebral blood flow.<sup>5</sup> ECT can also either increase or decrease the seizure threshold.<sup>6,7,8</sup> Nevertheless, the neurological side effect profile of ECT is reported to be minimal with most common symptoms being headache, disorientation, and memory complaints.<sup>9</sup> Whether ECT actually precipitated the cerebral embolism in this case is not clear. Change in cerebral blood flow or seizure threshold, which is a theoretical concern with

ECT, would not adequately explain the cause for this case. In the setting of normal sinus cardiac rhythm, the current literature does not support the idea of embolic event caused by ECT.

Our patient had a stroke that was most likely embolic in nature, considering its typical clot location and the stroke distribution on neurological imaging studies. In the absence of any identifiable causes of her stroke, this case could be classified as a cryptogenic stroke, and it is difficult to make any link between ECT and the embolic stroke especially in the setting of normal sinus rhythm. Clinicians should be aware that, even for patients with known AF, there is no report in the literature directly linking ECT with embolic stroke. In the rare event of stroke while receiving ECT, there is now a potentially effective treatment as demonstrated in this case. Careful neurological examination before, during, and after ECT would be helpful in diagnosing any adverse cerebral event that could be treatable.



**FIGURE 2.** A, Three-dimensional, multiprojectional, maximal intensity projection CT angiography images showing absence of filling in the right MCA distal M1 segment consistent with acute embolic occlusion. Axial image (A) and coronal image (B) are seen above.



**FIGURE 3.** A, Pretreatment angiography showing occlusion of the distal segment of M1. B, Successful IA rt-PA and wire lysis of the thrombus resulting in normal vessel caliber with normal distal flow.

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