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Case Report

Acute embolic stroke in a patient with atrial fibrillation after electroconvulsive therapy

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Summary Although patients with atrial fibrillation (AF) have an increased risk of embolic stroke, some clinicians hesitate to provide anticoagulation therapy for these patients during electroconvulsive therapy (ECT), which is widely applied for the treatment of intractable depression, bipolar disorder, and catatonic schizophrenia, because of potential intracerebral hemorrhage. We report on a 77-year-old female depressive patient with AF treated with aspirin but not on anticoagulation therapy because of poor compliance who developed embolic stroke 1 day after the last ECT. The $CHADS_2$ score of this patient was 2 and included the age and hypertension. The present case suggests that anticoagulation therapy should be considered for patients with obvious risks of embolic stroke when they are subjected to ECT.

Introduction

Electroconvulsive therapy (ECT) is widely performed against intractable depression, bipolar disorder, and catatonic schizophrenia. Although anticoagulation therapy is recommended for patients with atrial fibrillation (AF) to prevent embolic stroke before having ECT [1—3], some investigators consider anticoagulation as a factor increasing the risk of intracerebral hemorrhage during ECT [4] probably because ECT elicits a transient increase in blood pressure. As far as we know, there is no report demonstrating that embolic stroke developed after ECT in patients with AF. We report here a case with depression and AF with obvious risk factors of stroke but not on anticoagulation therapy with warfarin who developed embolic stroke after ECT.

Case report

A 77-year-old woman with chronic AF developed acute cerebral stroke 1 day after her 43rd ECT. The first ECT for this patient was operated 2 years before the onset of stroke because of severe depression refractory to medication therapy. She was admitted the Department of Psychiatry for gradual exacerbation of depressive symptoms 21 days before the stroke event. Her medications were aspirin, enalapril, and flunitrazepam. She had hypertension as a major risk

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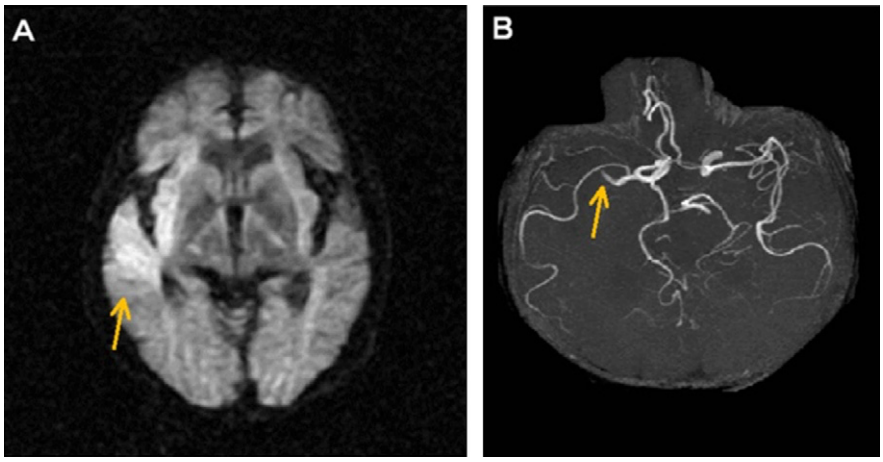

Figure 1 Magnetic resonance imaging at the onset of cerebral stroke. The right temporo-occipital lobe was highly diffused on diffusion-weighted magnetic resonance image (A). The upper trunk of the right middle cerebral artery was occluded on magnetic resonance angiography (B). Arrowheads indicate regions of interest.

factor of cerebral stroke, but had no history of cerebral ischemic events, diabetes, or heart failure. Although her $CHADS_2$ score [5] was 2, she had not had anticoagulation therapy with warfarin because of poor compliance. Transthoracic echocardiography performed 19 days before the onset of stroke revealed that left ventricular ejection fraction was 71% and there was no obvious thrombus although the left atrium was enlarged with a diameter of 5.3 cm and moderate aortic and mitral regurgitations were found. Her previous 42 ECTs had been all operated without any remarkable complications.

The 43rd ECT was safely operated at 14.00 h using the Thymatron System IV (Somatics, LLC, Lake Bluff, IL, USA) with brief-pulse square-wave currents. Before ECT was initiated, electrodes were placed in the traditional bilateral front-temporal manner, and atropin sulfate (0.25 mg) [6], landiolol (5 mg) [7], midazolam (10 mg), and succinylcholine (60 mg) were administered intravenously. Motor and electroencephalogram seizures lasting for 42 s and 56 s respectively were produced by the electrical stimulation. The parameters of ECT were as follows: electrical charge of 60% (297.2 millicoulombs [mC]), current of 0.88 A, stimulus duration of 6.7 s, frequency of 50 Hz, and pulse width of 0.50 ms. Flumazenil (0.3 mg) and nicardipine (0.5 mg) [8] were injected intravenously after the termination of seizure. Blood pressure and heart rate before ECT were 119/59 mmHg and 65 bpm respectively. The maximal blood pressure and heart rate during ECT were 145/83 mmHg and 83 bpm respectively. The heart rhythm was not converted to sinus rhythm during ECT. She was neurologically normal 1 h after the ECT.

At 13.00 h the next day, she suddenly fell down while she was walking in her room with right conjugate deviation of the eyes and left hemiplegia. Diffusion-weighted magnetic resonance imaging demonstrated diffusion in the right temporo-parietal region (Fig. 1A). Magnetic resonance angiography revealed occlusion of the upper trunk of the middle cerebral artery (Fig. 1B). She was diagnosed as having embolic stroke because of sudden onset of the symptoms and the existence of AF without anticoagulation therapy. After treatment with heparin, edarabone, glycerol, and mannitol her symptoms were rapidly relieved, and only slight muscle weakness remained in her left arm and leg 7 days after the onset of stroke.

Discussion

The patient with chronic AF developed acute cerebral stroke 1 day after ECT. To the best of our knowledge, there is only one report of thromboembolic stroke that was associated with ECT in a patient without AF [9]. Direct electrical influence of ECT on the heart has been reported to be negligible. Giltay et al. reported that intracardiac electrogram of a dual-chamber sensing VDDR pacemaker showed low noise signals of around 2 mV, which elicited oversensing in the atrium only during ECT [10]. Although arrhythmias such as atrial flutter and AF have been reported to develop after ECT probably due to the activation of parasympathetic tones in the early stage or that of sympathetic ones in the later stage [6,11,12], AF persisted during and after ECT in the present patient. Therefore, it is unlikely that the effect of ECT itself on electrical activity of the heart was responsible for the embolic stroke in the present case.

Embolic stroke is a well-known hazardous complication of AF. Therefore, anticoagulation therapy with warfarin is recommended for patients with a $CHADS_2$ index of 2 or more [13]. The index of the present patient was 2 because her condition satisfied age over 75 years and hypertension, both of which are definite risk factors of cerebral stroke.

Patients with depression have enhanced platelet reactivity and aggregation [14—17]. Some reports support the notion that depression itself is an independent risk factor of cerebral stroke [18]. Moreover, Stain-Malmgren et al. demonstrated that 5-H_2-receptor densities of platelets in patients with depression were increased after repeated ECT [19], which might enhance platelet reactivity and might increase the risk of embolic stroke. Therefore, the possibility of thrombotic events like cerebral stroke may be substantially higher in depressive patients subjected to ECT including the present patient than in ordinary AF patients. In summary, multi-factorial mechanisms, that is, no warfarin

use and enhanced reactivity and aggregation of platelets due to the existence of depression and repeated ECT may be responsible for the embolic stroke developed 1 day after the 43rd ECT in the present patient.

Anticoagulation therapy may increase the risk of intracerebral hemorrhage during ECT. As far as we know, however, only two cases with intracerebral bleeding were actually reported after ECT [20,21]. Furthermore, there is a report demonstrating that ECT in patients on anticoagulation therapy appears to be safe [22]. Considering the high annual stroke-event rate in patients with AF and obvious risk factors of cerebral stroke [13], it should be reasonable for these patients to have anticoagulation therapy especially when they undergo ECT for the management of depression.

Anticoagulation therapy with warfarin is recommended in a dose adjusted to achieve the target intensity international normal ratio (INR) of 2.0—3.0 for patients with AF at high risk of cerebral infarction [13]. However, the patient age and intensity of anticoagulation are powerful predictors of major bleeding. A lower target INR of 1.6—2.5 may be considered for primary prevention of cerebral stroke in patients with AF 75 years and older with increased risk of bleeding [13]. As we could not exclude the possibility that ECT is one of the risks of cerebral bleeding [20,21], the lower target intensity INR would be preferable especially in patients aged 75 years and older as in the case of the present patient.

When surgical procedures require interruption of oral anticoagulation therapy in patients with AF at high risk of cerebral infarction, unfractionated heparin or low-molecular-weight heparin may be given intravenously or subcutaneously, although the efficacy has not been established fully [13]. Whether the temporary discontinuation of warfarin and use of heparin should be considered in patients on anticoagulation therapy and requiring ECT remains to be elucidated [4].

Finally, transesophageal echocardiography is recommended for patients undergoing cardioversion who have not been on anticoagulation therapy [13]. It should also be clarified in future studies whether patients subjected to ECT without anticoagulation therapy should be screened for thrombus in the left atrium using transesophageal echocardiography.

Conclusion

Although there is a potential risk of intracerebral hemorrhage, anticoagulation therapy should be considered for patients with AF and obvious risks of cerebral stroke when they are subjected to ECT.

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