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Central pain associated with low thalamic blood flow treated by electroconvulsive therapy

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Introduction

Centrally acting interventions such as motor cortex stimulation therapy [1], electrical stimulation of the percentral cortical area [2], or electroconvulsive therapy (ECT) [3–5] have been reported to be effective in central poststroke pain in patients resistant to conventional therapies.

However, in previous clinical reports [3–5], the effects of ECT on regional cerebral blood flow (rCBF) were not studied. We present a case whose chronic central poststroke pain responded to a course of ECT treatment. To investigate the role of rCBF in ECT therapy, we measured thalamic blood flow using stable xenon-enhanced computed tomography (xenon-CT) [6] before and after a course of ECT.

Case report

A 58-year-old man suffered right-sided severe pain 6 months after a left thalamic hemorrhage. It was long-standing and intractable pain with dysesthesias, consisting of burning and throbbing throughout his right hemibody. It was resistant to conventional pain treatments such as stellate ganglion blocks, and various medications (intravenous ketamine, oral mexiletine, anticonvulsants, and antidepressants). Severe intractable central poststroke pain throughout his right hemibody, mostly in the upper and lower extremities, had lasted for almost 3 years.

Three years after the event, he was referred to the pain clinic of the Department of Anesthesiology, Shiga University of Medical Science, in order to receive a course of ECT treatment for his persistent pain. Visual analogue scale (VAS) levels for pain were rated as 8–10 (0, no pain; 10, maximal pain). He was unable to resume walking because of the persistent pain.

The patient was informed about the treatment protocol, which was approved by the Hospital Ethical Committee, and about the possible benefits and side-effects of ECT. Informed oral and written consent was obtained before ECT. Medication at that time consisted of 30 mg·day^{-1} nortriptyline and 300 mg·day^{-1} carbamazepin.

The ECT was conducted by brief-pulse stimulus (frequency 60 Hz, pulse width 1 ms, current 0.7 A, stimulus duration 2.8 s) once a week over 8 weeks unilaterally using a Thymatron DGX (Somatics) and seizure was assured by EEG monitoring and by viewing cuffed-limb seizures. The unilateral ECT application was to the nondominant hemisphere and ipsilateral to the cerebral vascular accident. Hypnosis was induced with a bolus injection of thiopental (2–3 mg·kg^{-1}), and muscle relaxation was achieved by succinylcholine (1–1.5 mg·kg^{-1} IV). Ventilation was assisted using a facemask with 100% oxygen, and nicardipine (0.5–1.5 mg IV) was injected to attenuate acute cardiovascular side-effects.

A course of eight unilateral ECT treatments resulted in a reduction in pain. After ECT treatment, the patient still had some occasional pain, but was able to tolerate it. VAS levels for pain after ECT were rated 2–5. His mental state and daily activities improved, and he could enjoy walking. He suffered retrograde and anterograde memory impairments transiently, but recovered within 2 months. One and half years after the ECT treatment, the reduction in central poststroke pain has persisted.

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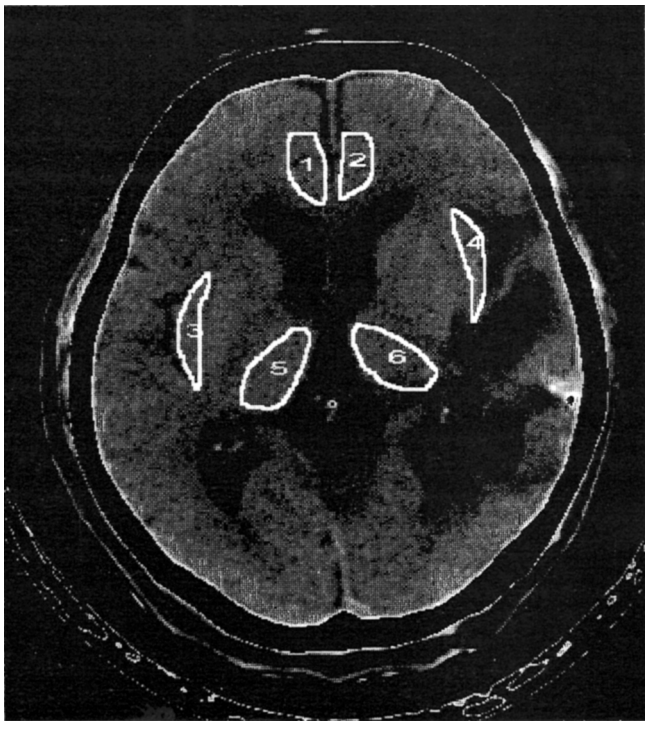

Fig. 1. Xenon computed tomography (CT) transverse section scan for regional cerebral blood flow (rCBF) measurement. We measured the rCBF of the thalamus before and after electroconvulsive therapy (ECT) using xenon-CT scans at a transverse CT slice that was 5 cm above the orbitomeatal line (OM-line) at the level of the basal ganglia. *1*,*2*, left and right subgeniculate part of the anterior cingulate cortex (Brodmann's areas 32), *3*,*4*, left and right insula, and *5*,*6*, left and right thalamus

To investigate the possible mechanisms of the analgesic effect of ECT, we measured the thalamic blood flow using xenon-CT before, and 7 days after, a course of unilateral ECT. rCBF values were measured using the conventional protocol (a wash-in, 5 min wash-out method with 3 min inhalation of 30% xenon gas) at a transverse slice 5 cm above the orbitomedial line from a single CT slice at the level of the basal ganglia, including the thalamus [6–8] (Fig. 1). rCBF calculations were semiautomatic, and done with Xe/CT software.

Prior to ECT, the mean rCBF in the right and left thalamus were 64.1 and 39.0 ($ml \cdot 100\,g^{-1} \cdot min^{-1}$), respectively. The thalamic blood flow contralateral to the side of the pain was significantly decreased. Before ECT, the left to right thalamic blood flow ratio was 61.0%. After ECT, the mean rCBF values of the right and left thalamus were 66.8 and 57.1 ($ml \cdot 100\,g^{-1} \cdot min^{-1}$), respectively, and the left to right thalamic blood flow ratio had risen to 85.0% when the pain subsided. The left thalamic blood flow after ECT increased by 46.0%.

Discussion

In this case, ECT alleviated chronic central poststroke pain. This contrasts with reports [8,9] which found a lack of response after a course of ECT in patients with thalamic pain. In these studies [8,9], the effects of ECT on rCBF were not studied. In our case, xenon-CT showed a significant decrease in rCBF in the thalamus contralateral to the side of the pain, and this decreased rCBF in the thalamus increased by 46.0% after ECT. The results from the xenon-CT suggest that the pain improvement was accompanied by an increase in the thalamic blood flow. The mechanism of the improvement in thalamic cerebral blood flow is not known at present. However, considering our xenon-CT data, it is suggested that the central poststroke pain threshold may be related to the thalamic blood flow.

Although it is unclear why ECT was so helpful in alleviating central poststroke pain in this case, recent reports suggest the following potential mechanisms for ECT's analgesic action. (1) ECT may block a pathological localized corticothalamic reverberatory loop which is involved in maintaining chronic neuropathic pain [10]. (2) ECT may activate inhibitory pathways via the activation of serotonergic, noradrenergic, and dopaminergic neurotransmission systems in the brain [11].

As compared with simple photon emission computed tomography (SPECT), xenon-CT used as a method for the measurement of rCBF offers a much higher spatial resolution, allowing a more precise reference of flow, and providing quantitative information on rCBF in deeper regions of the brain [7,8]. Furthermore, the values calculated by xenon-CT are quantitative, in contrast with those of SPECT, which produces only relative ratios [7,8].

In our patient, central poststroke pain relief after ECT was associated with an improvement in thalamic blood flow. This rCBF change may be related to the analgesic efficacy of ECT.

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