5. Drake ME Jr, Pakalnis A, Phillips B: Secondary mania after ventral pontine infarction. J Neuropsychiatry Clin Neurosci 1990; 2:322–325

Pathological Hyperfamiliarity for Others From a Left Anterior Cingulate Lesion

SIR: Person misidentification syndromes usually involve the misperception of familiar persons or a loss of familiarity for normally perceived ones. We report a unique patient who developed the opposite, a sense of hyperfamiliarity for unfamiliar persons after a hemorrhage in the left anterior cingulate cortex (ACC).

Case Report

A 34-year-old man developed an acute onset of a severe headache, followed by transient loss of alertness. He spontaneously recovered but 2 days later was hospitalized because of new-onset generalized seizures. The patient had a negative medical history and was on no medications at the time of admission.

On initial examination, he was in a postictal delirium. He had otherwise normal examinations of his cranial nerves, motor systems, and reflexes. He was loaded with phenytoin without further seizures. A computed tomography (CT) scan revealed a hyperdense region located in the left anterior cingulate cortex consistent with a hemorrhage. Angiography and magnetic resonance imaging (MRI) revealed a cavernous angioma. On electroencephalography, there were theta waves in the left frontocentral region.

One day after admission, mental status examination revealed an akinetic mute state. He was totally indifferent to external stimuli. Over the following days, the patient had gradually increasing verbal output and behavioral initiation. On day 7 after admission, neurobehavioral testing revealed normal orientation, attention, language, constructional abilities, calculation, reasoning, and judgment with impairment in verbal learning.

Surprisingly, the patient repeatedly expressed strong sensations of having previously known many of the hospital personnel. Sometimes he would ask if he knew them from school or his hometown, a rural village. The patient recognized that his feelings of familiarity were incorrect and strange but he continued to report the presence of these feelings. His sense of hyperfamiliarity for strangers gradually disappeared over a 2-week period, and he was discharged fully recovered.

Comment

The present case provides further evidence for the "assoziierende Erinnerungs- falschungen" phenomenon, or the sensation that unknown people are already known, originally described by Emil Kraepelin.[1] This person hyperfamiliarity syndrome is distinct from Fregoli's phenomenon, in which strangers are identified as familiar persons.[2] Fregoli patients change the personal identities of surrounding persons, but this patient only felt that they were familiar and did not change their identities.

This patient had a focal lesion in the left ACC, or Brodmann's Area 24. This corresponds to the "affect" region of the ACG, which regulates emotional awareness, as well as motivation and intention. Consistent with this localization, this patient had an initial akinetic muteness. His subsequent hyperfamiliarity syndrome implies that an alteration in emotional awareness for people is mediated by the ACC.

Prior investigations have suggested a right hemisphere person recognition network that links seen faces with representations of affective and personal relevance.[3–5] The right superior temporal gyrus, amygdala, and orbitofrontal cortex respond to facial emotions and contribute to a sense of familiarity from faces.[1–4] Hyperfamiliarity for unknown faces could arise from a hemispheric imbalance, with relative hypoactivation of left hemisphere processes but hyperactivation and spurious responsiveness of the right hemisphere person recognition network.[5]

Francisco Nente, M.D.
Neuropsychiatry Unit, National Institute of Neurology and Neurosurgery of Mexico
Roger Carrillo-Mezo, M.D.
Neuroimaging Department, National Institute of Neurology and Neurosurgery of Mexico
Mario F. Mendez, M.D.
Department of Neurology, University of California, Los Angeles, California
Jesus Ramirez-Bermudez, M.D., M.Sc.
Neuropsychiatry Unit, National Institute of Neurology and Neurosurgery of Mexico

References

1. Murai T, Kubota Y, Sengoku A: Unknown people believed to be known: the "assoziierende Erinnerungs- falschungen" by Kraepelin. Psychopathology 2000; 33:52–54
2. Dugall HS: Interictal psychosis presenting with Fregoli syndrome. J Neuropsychiatry Clin Neurosci 2004; 16:543–544
3. Mandal MK, Asthana HS, Maitra S: Right hemisphere damage impairs the ability to process emotional expressions of unusual faces. Behav Modif 1998; 22:167–176
4. Stone A, Valentine T: Perspectives on prosopagnosia and models of face recognition. Cortex 2003; 39:31–40
5. Vuilleumier P, Mohr C, Valenza N, et al:

Hyperfamiliarity for unknown faces after left lateral temporo-occipital venous infarction: a double dissociation with prosopagnosia. Brain 2003; 126:889–907

Topiramate Improves Glucose Metabolism in Choreatic and Depressive Patient: PET Findings

SIR: We measured regional cerebral glucose metabolism using 2-[18F]-fluoro-2-deoxy-D-glucose and positron emission tomography (FDG-PET) in a depressed patient with vascular hemichorea before and after treatment with a regimen of topiramate (25 mg/day, increasing to 100 mg/day over a 2-week period for 9 weeks). In order to reveal a defined information underlying the improvement of the depressive symptoms, we also combined the PET with Hamilton Depression Scale (HAMD). Although the striatal hypometabolic area was unchanged after 9 weeks of topiramate treatment, the right temporal area showed significantly increased glucose metabolism after treatment (1.58 vs 2.52). Interestingly, this increase was positively correlated with HAMD scores after 9 weeks of treatment (34 vs. 9). Beyond suggesting the efficacy of topiramate in vascular hemichorea, these findings suggest that the integrity of pathways linking the cortex and the basal ganglia together may be integral to the normal regulation of mood. The antidepressant efficacy which correlates to the increase in metabolism in neocortical regions further supports this finding.

Hemichorea-hemiballismus is a rare movement disorder which features involuntary movements of the limbs due to basal ganglia stroke being confined to the one side of the body.[1] Studies examining glucose metabolic rates or blood flow changes in regional brain areas have found that basal ganglia disorders are associated with decreased activity in the cortical regions.[2]

Topiramate is a broad-spectrum anticonvulsant. In addition to its therapeutic effect by vascular hemichorea-hemiballismus, topiramate was recently shown to be effective in reducing depressive symptoms in acute bipolar depression.[3]

Case Report

The patient, a 61-year-old man, experienced abrupt, involuntary movements of his left arm and left leg after he developed a right-sided stroke due to a basal ganglia infarction 6 months prior. In addition to these symptoms, he had developed in recent months a depressive mood; he felt sad, guilty, and lost practically all interest in doing things. The patient retreated from daily work activities and had difficulties with concentration and memory. He also described severe physical fatigue and moderate insomnia.

On psychiatric examination he was cooperative, alert, and fully oriented. He displayed a depressed mood state. His movements were slowed with signs of hypoactivity. During the examination, the patient cried and described morbid thoughts of death. The speech comprised simple vocabulary, then slowed and was spoken in a moderate depressive voice. It was sometimes interrupted by word-finding difficulties. The patient scored 34 on baseline HAMD.

A magnetic resonance imaging scan of the brain performed on the admission day showed a chronic lacunar infarct on the right lentiform nucleus on T2-weighted images correlating with the previous stroke 6 months prior. FDG-PET on admission day showed moderate to severe reduced glucose uptake right temporal cortex. Other cortical regions were found to be normal.

We started the patient on a regimen of topamirate 25 mg daily, increasing to 50 mg twice daily over a 2-week period. The optimum hemichorea control occurred when he was taking 50 mg of TPM twice daily after 2 weeks. A significant improvement of the depressive symptoms occurred, according to HAMD score, after 9 weeks of treatment. Interestingly, a PET follow-up after 9 weeks of topamirate treatment revealed more than a 70% (significant) increase in the right temporal area. In our study, the improvement of depressive symptoms seemed to be correlated with the normalized temporal metabolism after 9 weeks of treatment.

This was suggested by previous studies[4,5] which claimed that recovery from depression after antidepressive treatment was associated with a decrease of glucose metabolism in the limbic system and an increase in neocortical areas. Interestingly, this is the reverse pattern of normal mood regulation by which with depressive mood state, increases in limbic-paralimbic blood flow, and decreases in neocortical regions were identified.[6]

In addition to suggesting a functional interaction between the cortex and limbic system, our findings support that the integrity of pathways linking the cortex and the basal ganglia may be integral to the normal regulation of mood.

Burak Yulug, M.D.
Mustafa Bakar, M.D.
Department of Neurology, University of Uludag, Bursa, Turkey
Inanc Karapolat, M.D.
Department of Nuclear Medicine, Sifa Hospital, Izmir, Turkey
Orkide Güzel, M.D.
Department of Pediatric Neu-