Capgras Syndrome Related to Left-Hemisphere Injury

To the Editor: Capgras syndrome (CS) was first described in 1923[1] and is characterized by the belief that someone, often a close relative, has been replaced by an imposter. Although initially reported in schizophrenia, a substantial proportion of cases are related to neurologic pathology, noted almost invariably in the right hemisphere.[2] We present a patient who developed CS in the context of a left-hemisphere traumatic brain injury (TBI).

Case Report

A 72-year-old, right-handed man with no previous psychiatric or cognitive problems suffered a motorcycle accident resulting in intraparenchymal, subdural, and subarachnoid hemorrhages. He had a difficult hospitalization but ultimately returned home. Neuropsychiatric sequelae reportedly included memory and executive impairment and paranoia, the latter responding well to quetiapine. CT scan showed encephalomalacia limited to the left inferior frontal lobe.

About 2 months after discharge, he was seen in our clinic. His wife reported his having discontinued his quetiapine 1 month earlier because of worries about potential side effects. Since that time, she reported paranoid ideation about neighbors stealing from him, but also episodes of his misidentifying her as someone impersonating his wife in order to steal. During these episodes, the patient would become agitated, but would generally respond to reassurance. He was oriented to person and place but not to date, and denied the episodes of misidentification. He often perseverated about his sailing experience in the Navy.

Over the next 5 months, the episodes of misidentification continued; his wife ultimately agreed to a trial of risperidone, but he again quickly stopped the medication because of worries about potential side effect. At the last visit, she reported the episodes of misidentification as gradually decreasing and responding better to reassurance. She declined any further medication trials.

CS has been reported frequently in neurodegenerative diseases, stroke, and TBI, and the right hemisphere has generally been implicated in its genesis.[2] Our patient's injury, however, was to the left frontal lobe, and it is difficult to reconcile this with previously reported cases. Several potential explanations come to mind. Although neuroimaging showed encephalomalacia limited exclusively to the left frontal lobe, there may conceivably have been a covert right-hemisphere contrecoup injury, as well; possibly, an MRI scan would have been helpful.

Alternatively, Joseph[3] has hypothesized that CS and other misidentification syndromes may arise from a disconnection between right and left cortical areas that decode afferent sensory information. Finally, seizure disorders are common complications of TBI and may be associated with delusions and other psychiatric symptoms,[4] although an association between CS and seizures has not been suggested. Unfortunately, no EEG was performed. Our patient's CS seemed to improve at least modestly with time; it is not clear what his course would have been with consistent antipsychotic treatment.

Our understanding of the neuropathology of CS remains limited. This case suggests that CS after left hemisphere damage may occasionally occur, although potential mechanisms are elusive.

This work was presented at the American Neuropsychiatric Association annual meeting in Tampa, FL, March 2010.

Jean M. Fils, M.D.
Jonathan T. Stewart, M.D.
James A Haley VA Hospital, University of South Florida College of Medicine, Tampa, FL
Correspondence: jonathan.stewart1@va.gov

References

1. Capgras J, Reboul-Lachaux J: L'illusion des "sosies" dans un delire systematise chronique. Bull Soc Clin Med Ment 1923; 11:6–16
2. Feineberg TE, Roane DM: Delusional misidentification. Psychiatr Clin North Am 2005; 28:665–683
3. Joseph AB: Focal central nervous system abnormalities in patients with misidentification syndromes. Bibl Psychiatry 1986; 164:68–79
4. Fujii D, Ahmed I: Characteristics of psychotic disorder due to traumatic brain injury: an analysis of case studies in the literature. J Neuropsychiatry Clin Neurosci 2002; 14:130–140