

# MULTIPLE FREGOLI DELUSIONS AFTER TRAUMATIC BRAIN INJURY

Todd E. Feinberg<sup>1</sup>, Lisa A. Eaton<sup>2</sup>, David M. Roane<sup>3</sup> and Joseph T. Giacino<sup>2</sup>

(<sup>1</sup>Yarmon Neurobehavior and Alzheimer's Disease Center, Beth Israel Medical Center, New York; <sup>2</sup> JFK Johnson Rehabilitation Institute, Department of Neuropsychology, Edison, New Jersey; <sup>3</sup>Beth Israel Medical Center, Department of Psychiatry, New York)

## ABSTRACT

A 61 year old man after a traumatic brain injury resulting in right frontal and left temporoparietal contusions developed florid Fregoli-type misidentifications. Extensive neuropsychological testing demonstrated significant deficits in executive and memory functions. The patient's neuropsychological profile closely resembled that seen in previously reported patients with Capgras syndrome. Our findings are consistent with the hypothesis that a combination of executive and memory deficits may account for cases of delusional misidentification associated with brain lesions. However, the form which the delusion takes may be influenced by other factors including motivation.

Key words: Fregoli syndrome, misidentification.

## INTRODUCTION

In 1927, Courbon and Fail described a 27 year old woman with schizophrenia who believed that famous actresses, family members, and persecutors were appearing in the form of acquaintances, strangers, and hospital staff. They referred to this condition as the Syndrome d'illusion de Fregoli after an actor who was famous for his impersonations. The Fregoli syndrome is one of a number of disorders known as delusional misidentification syndromes (DMS) along with the Capgras syndrome (Capgras and Reboul-Lachaux, 1923), the syndrome of intermetamorphosis (Courbon and Tusques, 1932), the delusion of subjective doubles (Christodoulou, 1978) and reduplicative paramnesia (Pick, 1903). In all of these, a patient incorrectly identifies or reduplicates persons, places, objects, or events (Feinberg and Shapiro, 1989; Feinberg and Roane, 1997a).

While Capgras syndrome has traditionally been associated with psychiatric illness, there has been increasing recognition that Capgras occurs with neurological diseases such as Alzheimer's disease (Mendez, Martin and Smyth, 1992), head trauma (Alexander, Stuss and Benson, 1979), cerebrovascular disease (Forstl, Almeida, Owen et al., 1991), epilepsy (Forstl et al., 1991) and Parkinson's disease (Roane, Rogers, Robinson et al., 1998). To a lesser extent, the Fregoli syndrome has also been linked to neurological dysfunction. Christodoulou (1976) found EEG abnormalities in 7 consecutive Fregoli patients,

including 6 diagnosed with schizophrenia. Joseph and O'Leary (1987) found greater frontal and temporal atrophy in 10 patients with Fregoli syndrome than in 10 matched controls. De Pauw, Szulecka and Poltock (1987) described a patient who developed the Fregoli syndrome after a right temporo-parietal lobe infarct, but this patient had a previous history of psychiatric treatment and paranoid symptoms.

Given the overlap between neurological and psychiatric causation of DMS, it is important to analyze cases in which no preexisting psychiatric pathology is in evidence and to obtain adequate neuropsychological data to determine the integrity of underlying neurocognitive functions. We had the opportunity to examine a patient with no premorbid psychiatric history who developed florid Fregoli-type misidentifications following a traumatic brain injury. We conducted extensive structured interviews with the patient and his wife and completed neuropsychological assessment during the period of Fregoli misidentification. This was done in an effort to correlate the phenomenologic features of Fregoli syndrome with the cognitive and neuropathologic substrates.

#### HISTORY, BACKGROUND INFORMATION AND ANATOMIC ANALYSIS

BJ is a 61 year old right handed man who sustained a moderate to severe closed head injury following a fall down a flight of stairs leading to his basement. Prior to the injury, he was employed as a supervisor for a large communications company and had a forty year work history. He has been married for forty-four years and has two adult children. He was a high school graduate. The past medical history is significant for hypertension and alcohol use but no evidence of significant disturbance in functional capacity.

On the day of the injury, he was found by his wife at the bottom of the basement stairs. He was reportedly muttering a few words but was otherwise unresponsive to verbal prompts. He was combative en route to the hospital and was given a Glasgow Coma Scale score of 10 on admission to the emergency room. Neurologic examination found "raccoon eyes", left hemotympani, sluggish pupillary reactivity on the left and mild left hemiparesis. Lab studies showed an elevated blood alcohol level. The initial CT scan (Figure 1) showed, in addition to a left parietal depressed skull fracture, large right frontal and smaller left temporoparietal contusions with surrounding edema and encephalomalacia.

Progressive improvement in mental status was noted across the acute course and BJ was transferred for inpatient rehabilitation on day 16.

#### NEUROPSYCHOMETRIC FINDINGS

Formal neuropsychological assessment was completed approximately three months from the date of injury. At the time of the evaluation, BJ was generally compliant although periods of irritability were noted. He was oriented to person and year but mis-stated the name and location of the hospital. Spontaneous speech was logorrheic and responses to questions regarding circumstances

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