## Interictal Psychosis Presenting With Fregoli Syndrome

*SIR:* Interictal psychosis in patients with epilepsy is highly correlated with temporal lobe epilepsy.[1] Fregoli syndrome is the delusional misidentification of familiar persons disguised as others. While the organic etiology of Capgras syndrome, another delusional misidentification syndrome (DMS), is widely studied, there are only anecdotal reports on a similar etiology for Fregoli syndrome. A case of interictal psychosis presenting with Fregoli syndrome is described and the neurobiological basis of the syndrome is discussed.

**Case Report**

A 30-year-old woman was admitted with first break psychosis of acute onset characterized by disorganized behavior, loosening of association, auditory hallucinations, and persecutory delusions. The striking feature of the patient's psychopathology was her misidentifying strangers as people known to her. In the emergency room, she misidentified another patient as her boyfriend and on the unit she misidentified the nurse as her mother and the social worker as her sister. Though these persons bore no close resemblance with the patient's family members, the patient was convinced that her family members had been transformed into these strangers.

The patient had no prior psychiatric history and family history was noncontributory. Following a motor vehicle accident 8 years ago, she was being treated with oxcarbazepine (600 mg twice a day) for complex partial seizures with secondary generalization. Last episodes of generalized tonic clonic seizure and complex partial seizure were 2 years and one month ago, respectively. Her neurological examination and laboratory work up was unremarkable. A Mini-Mental State Examination was attempted but the patient's severe disorganization precluded any meaningful interpretation. While in the hospital, her initial EEG was reported as normal while another one done two weeks later when the psychosis started to remit showed left temporal spike and sharp waves. The MRI of the brain was normal. The patient was treated with risperidone and her oxcarbazepine was increased to 900 mg twice a day. Her psychosis, including the delusions of misidentification, completely resolved within a month and a repeat EEG done before the discharge did not show any epileptiform discharges. Interestingly, the patient had no memory of her misidentifying the staff on the unit.

**Comment**

The notable feature of this presentation was the occurrence of Fregoli syndrome in the setting of epilepsy. EEG abnormalities and epilepsy have been anecdotally reported with Fregoli syndrome.[2] Other conditions associated with Fregoli syndrome include stroke, head injury and Alzheimer's dementia.[2,3] One study demonstrated anterior cortical atrophy in psychotic patients with Fregoli syndrome compared to those without this syndrome.[4] Although the bulk of literature on DMS implicates a right-sided brain pathology,[2] interictal psychosis per se is most commonly associated with a left-sided functional brain abnormality.[1]

The neurobiological basis of Fregoli syndrome is discussed below. Capgras syndrome, the commonest of the DMS involving "hypo-identification," is understood to result from right temporolimbic-frontal disconnection resulting in a disturbance in familiarity of people and places.[3] However, this hypothesis may not explain the Fregoli syndrome, which involves "hyper-identification." Thus alternative hypotheses have been explored and one such hypothesis postulates that interhemispheric disconnection of cortical areas could allow each hemisphere to establish independent images of a person, place or event.[3] Hence if one hemisphere cannot explain the perception received by the other hemisphere, the individual deals with this discrepancy by confabulating about the experience. This has been demonstrated by split-brain research when subjects with callosotomy confabulate on presenting with different visual stimuli to either hemisphere.[5] Interestingly, some investigators regard DMS as a type of confabulation citing evidence from Korsakoff and Hughlings Jackson.[3] In this context, DMS is a disturbance in personal relatedness between the self and environment and Fregoli syndrome entails an insertion of personal relatedness (confabulation) resulting in "overpersonalized misidentification." Finally, frequent seizures may lead to aberrant plastic regeneration of synapses,[1] with the resultant "miswiring" leading to a functional disconnection between the cortical areas. That carbamazepine, an antiepileptic that inhibits kindling in temporal lobe, has been

helpful in treating individual cases of DMS supports the above notion.[6]

HARPREET S. DUGGAL, M.D., D.P.M.
Western Psychiatric Institute and Clinic, Pittsburg, PA

**References**

1. Sachdev P: Schizophrenia-like psychosis and epilepsy: the status of the association. Am J Psychiatry 1998; 155:325–336
2. Mojtabai R: Fregoli syndrome. Aust NZ J Psychiatry 1994; 28:458–462
3. Feinberg TE, Eaton LA, Roane DM, et al: Multiple Fregoli delusions after brain injury. Cortex 1999; 35:373–387.
4. Joseph AB, O'Leary DH: Anterior cortical atrophy in Fregoli syndrome. J Clin Psychiatry 1987; 48:409–411
5. Baynes K, Gazzaniga MS: Consciousness, introspection and the split-brain: the two minds/one body problem, in The New Cognitive Neurosciences, 2nd ed. Edited by Gazzaniga MS. Cambridge, Mass, MIT Press, 2000, pp 1355–1363
6. DePauw KW, Szulecka TK, Poltock TL: Fregoli syndrome after cerebral infarction. J Nerv Ment Dis 1987; 175:433–438

## Ejaculation After Defecation Without Orgasm Induced by Milnacipran

*SIR:* Milnacipran is a novel serotonin (5-hydroxytryptamine [5-HT]) and noradrenaline reuptake inhibitor (SNRI). To our knowledge, this is the first report of a depressive patient who experienced ejaculation after defecation without orgasm in the treatment of milnacipran.

**Case Report**

Mr. A was a 31-year-old man. He had no past and familial history of psychiatric and urologic disorders. He got depressed and started to have milnacipran of 50 mg/day. One week later, the daily dosage of milnacipran was increased to 100 mg. Four weeks after the beginning of milnacipran, he first experienced ejaculation after defecation without orgasm. As he felt ashamed of having such a symptom, he did not report it to the doctor. Seven weeks after the beginning of milnacipran, his depressive symptoms remitted. At that time he reported his ejaculation after defecation to the doctor. He wished to keep on having milnacipran in spite of that symptom, because he was afraid of a recurrence of depression. Milnacipran was prescribed thereafter. He continued to experience ejaculation after defecation about once per 2 or 3 weeks. Additionally, he occasionally observed emission of sperm at the end of micturition. One year after his first visit to our hospital, milnacipran was gradually decreased and stopped. He no longer experienced ejaculation after defecation. He started his new sexual life about 3 months after the cessation of milnacipran. He had no sexual dysfunction.

**Comments**

He had no intercourse for about 6 months before the beginning and for about 3 months after the cessation of milnacipran; nevertheless he did not experience ejaculation after defecation and emission of sperm at the end of micturition during those periods. This course shows that involuntary ejaculation was caused by the medication of milnacipran, not by the overpooling of sperm.

5-HT acts inhibitory on seminal ejaculation in the brain.[1] The sexual side effects most frequently observed in selective 5-HT reuptake inhibitors (SSRIs) are delayed ejaculation and absent or delayed orgasm.[2] 5-HT increased by SSRIs is supposed to cause these side effects by its central effect. On the other hand, peripherally, seminal emission and ejaculation are primarly under adrenergic control.[3] There have been two case reports about spontaneous ejaculation induced by reboxetine.[4,5] Reboxetine is a selective noradrenaline reuptake inhibitor, and noradrenaline increased by reboxetine is supposed to cause spontaneous ejaculation by its peripheral effect. Though venlafaxine, one of SNRIs, showed ejaculation dysfunction in the frequency of 12% in a study of 1,033 patients, milnacipran was not reported to show ejaculation disturbance in a study of 1,867 patients.[6] It might be due to well-balanced inhibitory effect of milnacipran on the reuptake of 5-HT and noradrenaline. However, this case suggests that milnacipran also has a possibility to induce ejaculation dysfunction. Patients generally hesitate to talk about their sexual problems with the doctor; therefore, this kind of side effects can be underestimated. We should be careful about the sexual dysfunction induced by milnacipran.

KEIZO YOSHIDA, M.D., PH.D.
HISASHI HIGUCHI, M.D., PH.D.
HITOSHI TAKAHASHI, M.D., PH.D.
TETSUO SHIMIZU, M.D., PH.D.
Department of Psychiatry, Akita University School of Medicine, Akita, Japan

**References**

1. McIntosh TK, Barfield RJ: Brain monoaminergic control of male reproductive behavior, I: serotonin and the post-ejaculatory refractory period. Behav Brain Res 1984; 12:255–265
2. Rosen RC, Lane RM, Menza M: Effects of SSRIs on sexual function: a critical review. J Clin Psychopharmacol 1999; 19:67–85
3. Gutierrez MA, Stimmel GL: Management of and counseling for psychotropic drug-induced sexual dysfunction. Pharmacotherapy 1999; 19:823–831
4. Haberfellner EM: Sexual dysfunction caused by reboxetine. Pharmacopsychiatry 2002; 35:77–78
5. O'Flynn R, Michael A: Reboxetine-induced spontaneous ejaculation. Br J Psychiatry 2000; 177:567–568
6. Briley M: Specific serotonin and noradrenaline reuptake inhibitors (SNRIs): a review of their pharmacology, clinical efficacy and tolerability. Hum Psychopharmacol 1998; 13:99–111