

### Response of Catatonic Schizophrenia to Amisulpride: A Case Report

Dear Editor:

Catatonia is a neuropsychiatric syndrome in which an abnormal mental state is associated with cataleptic phenomena; namely, akinesia, posturing, and mannerisms (1). Despite its emerging link to affective disorders, catatonia is still classified in DSM-IV as a subtype of schizophrenia. The prevalence is as high as 10% of all schizophrenia cases (2). Catatonia can be acute or chronic; therefore, treatment varies accordingly (3). Acute presentations are most effectively treated with high dosages of benzodiazepenes or with ECT (4,5). Case reports of treatment with antipsychotics exist (6,7). Chronic presentations prove more challenging to treat and have shown poor response to these standard therapies (3). We report a case of an individual with chronic catatonic schizophrenia who responded to amisulpride. To our knowledge, this is the first reported case of its kind in the literature.

#### Case Report

Mr D, a 24-year-old white man, was hospitalized for catatonic schizophrenia at age 21 years. His symptoms included mutism, facial grimacing, posturing, and periods of catatonic excitement and stupor. Extensive laboratory evaluations and diagnostic imaging were all unremarkable. Scores on the Bush-Francis Catatonia Rating Scale (BFCRS) varied throughout his illness, with a high of 18. Adequate trials of lorazepam, ECT, loxapine, and olanzapine yielded minimal response. A trial of clozapine was undertaken with some symptom reduction and later augmented with lamotrigine. Subsequent neutropenia developed, leading to discontinuation of

both drugs. The patient was started on risperidone and titrated to 6 mg daily in conjunction with lorazepam 6 mg daily. This combination produced moderate improvement after 2 months; however, for unclear reasons, the patient began refusing lorazepam. Significant extrapyramidal side effects (EPSE) developed, which necessitated a reduction in risperidone dosage, and subsequent clinical deterioration ensued. Augmentation with gabapentin and quetiapine proved ineffective. Amisulpride was then added to risperidone and titrated to 600 mg daily. After 2 months on this combination therapy, the patient began to engage in nonverbal communication. He achieved complete resolution of mutism after another 3 weeks. By the third month of amisulpride, all catatonic symptoms had resolved. The patient was discharged 6 months later on amisulpride 300 mg twice daily and risperidone 1.75 mg daily. His BFCRS score on discharge was 0.

#### Discussion

Catatonia is thought to result from a hypodopaminergic state (8) but may also involve dysfunction in neurotransmission of GABA and glutamine (4). The use of antipsychotics in treating catatonic schizophrenia is controversial, because the therapeutic effectiveness is exerted through dopamine antagonism. In fact, catatonia is cited as a risk factor for developing neuroleptic malignant syndrome (NMS) (5). The acute catatonia in this individual did not respond to first-line agents (benzodiazepenes and ECT), leading to untreated illness and chronicity. Chronic catatonia may involve a different pathophysiologic mechanism of action than the acute counterpart (3), advocating for the use of antipsychotics to target the underlying psychotic illness. In our case,

risperidone provided partial response and was maintained for this reason. Adding amisulpride alleviated Mr D's catatonic symptoms dramatically. It remains to be determined, however, whether symptom resolution was attributable to amisulpride alone or to the combined therapy. Because receptor profiles in the 2 drugs differ, a degree of synergy may be in operation (9,10). This result may allow for further elucidation of the pathophysiologic mechanism of chronic catatonic schizophrenia and promote the judicious use of atypical antipsychotics in its treatment.

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## Medication Noncompliance Among Psychiatric Outpatients In Iran

Dear Editor:

Throughout the world, medication non-compliance is one of the major problems in psychiatric treatment. We have experienced this obstacle almost daily in our outpatient clinics in Iran. Medication noncompliance is defined as discontinuation of medications without physician recommendation. We should make clinicians aware that medication noncompliance is common and may be a relatively unpredictable phenomenon. Further, it can become a major treatment difficulty in psychiatric patients. We conducted a study in Kerman, Iran, which assessed 150 randomly selected psychiatric outpatients for medication noncompliance. Of the subjects, 56 (37.4%) had a history of early discontinuation of psychotropic medications. Medication noncompliance was more frequent in patients with primary school education than in those with high school education. False beliefs about psychotropic medications were significantly higher in patients who discontinued their treatment before the appointed time than in patients who continued.

The most prevalent false belief is that psychotropic medications are addictive. According to diagnosis, the highest rate of early discontinuation was seen among patients with depressive disorders. A high rate of medication noncompliance was also found in other studies (1,2).

Hopelessness, an important symptom, may be responsible for medication non-compliance in patients with depressive disorders. A false belief in psychotropic medications is a contributing factor to medication noncompliance in our country.

Patient compliance is an important factor for better therapeutic success; thus, for improved outcome, we recommend that psychiatrists evaluate patient compliance

before prescribing psychotropic medications.

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## Diogenes Syndrome in a Pair of Siblings

Dear Editor:

Psychotic disorders or severe cognitive impairment are the usual causes of extreme self-neglect and social withdrawal when occurring in elderly subjects who have been socially efficient. Such behaviours, however, have been reported in individuals without any psychotic troubles or dementia (1). The denomination of this syndrome was suggested by Clark and others, inspired by the 4th century BCE ascetic Greek philosopher, Diogenes, who advocated the principles of self-sufficiency, freedom from social restraint, lack of shame, and rejection of material values (2). Diogenes syndrome (DS) is characterized by severe self-neglect, domestic squalor, social withdrawal, stubborn refusal of any well-meant help, and sometimes a tendency to hoard rubbish (sylllogomania) (3). Although most reported DS occurs in subjects who live alone, 2 cases have been described in married couples (4,5). We present the first case report, to our knowledge, of DS in a pair of siblings.

### Case Report

Miss M, a 61-year-old unmarried woman, and her 58-year-old handicapped brother were visited in their flat by the local mental health service at the request of their neighbours, who complained about an intolerable smell. Mr M's forearms had

been amputated at age 11 years after a serious accidental electrocution. For at least 1 year, he persistently refused artificial limbs and surgery for osteoarthritis in his hip bones; hence, he was obliged to remain lying on his bed. Their mailbox was filled to the brim, and because they didn't pay invoices, electricity had been switched off.

At the entrance, the flat was grossly dirty and untidy, with an unbearable stench. Rubbish had been hoarded to the extent that most of their living space was taken up with full cardboard boxes, bins, and heaps of magazines. Miss and Mr M denied the precariousness and the insanitariness of their living conditions and vehemently refused any assistance. For this reason, hospitalization in the psychiatric department was decided for both.

Contrasting with their isolation and the flat's filthiness, and in spite of his handicap, Mr M was in good health, without bedsores, anemia, or other biological signs of malnutrition. Cognitive and psychiatric assessments did not reveal any dementia, cognitive impairment, or psychotic disorders in either patient. During the hospitalization, Miss and Mr M minimized the seriousness of the flat's damage, and similar personality traits were noted: they were both aloof, secretive, and suspicious. Finally, we diagnosed a primary DS.

### Discussion

DS cases reported in the literature refer to various and heterogeneous conditions. Most DS subjects live alone and are single or widowed (1). DS in a pair of subjects is a rare condition, with few cases reported in the literature, all concerning married couples (1,4,5). To our knowledge, this is the first reported case of a DS occurring in a pair of siblings.

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## Treatment Option for Seasonal Affective Disorder

*Dear Editor:*

The following presentation revisits the diagnosis and successful treatment of major depressive disorder (MDE) with seasonal pattern.

### Case Report

Mr KC, a 29-year-old African-American married man, has a 6-year history of apathy, low energy, anhedonia, sadness, hopelessness, insomnia, and increase in joint pain, leading to impairment in his performance of military duties, as well as in his relationship with his wife. Onset of symptoms occurred yearly, beginning in the fall and remitting in the spring. A detailed history revealed no symptoms of mania, psychosis, or anxiety. He has no history of alcohol and drug use. A family history revealed a biological mother suffering from major depression and a biological father suffering with alcoholism. Other than knee-joint pain, the patient has good physical health. He has been employed with the military for over 10 years. He presented to his physician at a military base with these symptoms and was diagnosed with major depression with seasonal pattern. Phototherapy treatment was unavailable at the military base. Further, because of duty shifts, it could not be offered as an option. A review of antidepressants provided evidence of a trial of selective serotonin reuptake inhibitors (SSRIs). Thus, a trial of citalopram 20 mg daily

was initiated. At 2 and 4 weeks follow-up, Mr KC reported an increase in his energy level, an upbeat feeling, an improvement in mood, a restoration of normal sleep, and a significant decrease in knee-joint pain. His wife provided collateral information confirming this improvement. He started using less nonsteroidal antiinflammatory drug (NSAID) medication for pain and could return to some athletic activities, which he could not tolerate earlier.

### Discussion

The DSM-IV states that the regular temporal relation between the onset of major depression in fall or winter and its full remission in spring characterizes seasonal patterns. In Alaska, a study estimated a prevalence of 9.2% for seasonal affective disorder (SAD) (1). Light therapy has been the treatment of choice, with minimal side effects. Headache, eyestrain, and harmful effects on the retina are, however, potential side effects (2). Data for the pharmacologic treatment of SAD are rather limited. In controlled trials, however, fluoxetine and propranolol have been effective. Open trials have also shown positive results with bupropion and monoamine oxidase inhibitors (3,4). One National Institute of Mental Health group has studied response to the serotonergic agent, m-cpp, in SAD patients, suggesting that serotonin dysregulation is an area for further study. One case study suggested that citalopram, an SSRI, was as effective as phototherapy for SAD. Because the neurobiology and the diagnostic validity of SAD is not always obvious, good history taking and a trial of antidepressants, such as citalopram, may prove to be an effective treatment option alone (5,6).

One major question that remains to be studied is whether patients with SAD should be weaned off antidepressants in the spring. If successful weaning off occurs, the same antidepressant

medication may be as effective for the next likely episode.

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## Factitious Anemia and Magnetic Resonance Imaging Abnormalities

*Dear Editor:*

Little is known about the causes and pathophysiology of factitious disorders. Several authors have proposed the possibility of preexisting organic cerebral lesions. Frontotemporal cerebral atrophy (1), marked hyperperfusion of the right hemithalamus (2), cerebral palsy (3), or areas of high signal in periventricular white matter in both hemispheres in T2-weighted magnetic resonance imaging (MRI) (4) have been respectively found in 4 patients who displayed Munchausen syndrome. We report a case of factitious anemia with MRI abnormalities, similar to those described by Fenelon and others (4).

### Case Report

Ms O, a 35-year-old unmarried woman, was admitted to our psychiatric department after a suicide attempt. At psychiatric examination, Ms O did not fulfill DSM-IV criteria for any psychotic or affective disorder, and the suicide

attempt rapidly appeared to be a response to perceived rejection. Throughout her hospitalization, Mrs O exhibited irritability, suggestibility, and poor insight.

A review of her medical record indicated a psychiatric history of factitious anemia that began at age 15 years and included many explorations and hospitalizations for 13 years. At age 28 years, she complained of transitory neurological symptoms, such as visual disturbances, paresthesia, and dizziness. Even so, the successive neurological examinations and complementary examinations (cerebrospinal fluid analysis, central visual field, and auditory, visual, and somatosensory-evoked potentials) were normal. The MRI of the brain, however, showed areas of high signal in periventricular white matter in both hemispheres in the T2-weighted sequence. These abnormalities, although not specific, first suggested the possibility of multiple sclerosis. Nevertheless, the normality of the different neurological examinations and the performed explorations did not favour this diagnosis.

## Discussion

To our knowledge, this is the first report of MRI abnormalities in a patient with a factitious anemia. The first question raised is the link between the neurological symptoms and the MRI abnormalities. Although the diagnosis of multiple sclerosis cannot be invalidated, the absence of other objective neurological abnormalities does not support it. In this context, the subjective neurological complaints reported by the patient are consistent with the hypothesis of a neurological factitious disorder. The second remaining question is whether the MRI abnormalities that were found played a role in the development of factitious anemia.

This report raises an interesting question about cerebral lesions in the occurrence of pathomimia. To illuminate this association, we suggest further investigation to

increase our understanding of the pathophysiology of factitious disorders.

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## Pregnancy and Respiratory Nocturnal Panic Attacks

*Dear Editor:*

Pregnancy may have a highly variable influence on the course of panic disorder (PD) (1). Some case reports suggest that pregnancy protects against PD (2), but worsening or no change of PD during pregnancy is also reported (1).

We describe 2 women who were referred to the Laboratory of Panic and Respiration of the Federal University of Rio de Janeiro, with PD with prominent respiratory nocturnal panic attacks during pregnancy only.

### Case Report 1

Mrs A, a 30-year-old white woman, started at age 26 years to have spontaneous diurnal attacks with palpitations, shortness of breath, choking, chest pain, dizziness, and fear of losing control. She was treated with imipramine (150 mg daily). After 18 months, she was asymptomatic and started to decrease the dosage. She had her first pregnancy after being free of psychotropic medications for 1 year. During the fourth month of pregnancy, however, she developed recurrent panic attacks during sleep. Her

nocturnal panic attacks were intense and occurred almost nightly, with prominent respiratory symptoms (shortness of breath, chest pain, tingling, fear of dying and losing control, and severe choking). She never experienced nocturnal panic attacks before her pregnancy. She developed agoraphobic behaviour; she could only sleep seated, given her intense anticipatory anxiety at nightfall. She was initially treated with nortriptyline 10 mg daily. After 6 weeks, at the dosage of 75 mg daily, she achieved full remission of her respiratory nocturnal panic attacks. During her pregnancy, there was no diurnal panic attack. During the last 2 weeks of her pregnancy, nortriptyline was no longer prescribed. The asymptomatic period persisted after a 2-year follow-up.

### Case Report 2

Mrs B, a 32-year-old white woman, began having diurnal panic attacks at age 22 years. During her panic attacks, the main symptoms were choking and shortness of breath. She was treated with paroxetine 20 mg daily. After 2 months, she no longer had panic attacks. For the next 32 months, she remained asymptomatic. During the fifth month of her second pregnancy, however, she started experiencing nocturnal panic attacks, with shortness of breath, chest pain, dyspnea, choking, paresthesias, sweating, tachycardia, and severe fear of dying. There was an intense anticipatory anxiety during sleep. She developed fear in falling asleep and performed her usual daily activities during the night. She was treated with nortriptyline and a dosage of 75 mg daily achieved complete remission. The drug was gradually discontinued during the last month of her pregnancy. At her 3-year follow-up, she had been without any panic attacks.

## Discussion

The respiratory PD subtype appears as a distinct sample (3). Nocturnal panic attacks are common, often neglected, and have been presenting with important respiratory symptoms. The physiology of

the sleep, characterized by a significant rise of blood CO<sub>2</sub> (4), may explain why nighttime is favourable for developing panic attacks in patients with PD, a population characterized by heightened CO<sub>2</sub> sensitivity (4). Sex hormones influence respiration, and pregnancy is characterized by a strong physiologic fluctuation of their blood levels. The rise of progesterone levels may protect against panic attacks by facilitating GABA-ergic activity and by diminishing arterial CO<sub>2</sub> levels through increased minute ventilation (5); however, synthetic progestones can cause severe dyspnea, tachypnea, and hyperventilation (6), and estrogens can induce panic attacks (7). We can conclude that, during pregnancy

in some patients with PD, the balance among sex hormones may negatively affect respiratory sensitivity, thus inducing panic attacks. In addition, during nighttime, the pregnant woman's sleeping position may obstruct diaphragmatic breathing, modifying respiratory patterns to the extent that they enhance the possibility of nocturnal panic attacks.

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