Organic Hypomania Secondary to Sibutramine-Citalopram Interaction

Sir: Sibutramine, a serotonin-norepinephrine reuptake inhibitor (SNRI), is marketed for weight loss and weight maintenance.[1] I report a unipolar depressed woman who had hypomania induced by sibutramine in combination with citalopram. No similar reports were found on MEDLINE.

Case report. A 43-year-old woman presented for treatment of major depressive episode with atypical features, based on the Structured Clinical Interview for DSM-IV (SCID).[2] She had had unipolar major depressive disorder since age 35, and the present episode had lasted for 2 years. She had been taking citalopram, 40 mg daily, for 2 years, and she had not responded to fluoxetine, 60 mg daily, added to citalopram months before. Because she had become obese, she had been given sibutramine (standard 10-mg daily dose) by the treating psychiatrist a few weeks before, while still taking citalopram. During the interview, she said that a few hours after the first sibutramine dose, she had experienced irritability, insomnia, more talkativeness, racing thoughts, distractibility, hyperactivity, psychomotor agitation, shivering, and diaphoresis, without marked impairment of functioning. These symptoms had persisted for 3 days during the period she had continued sibutramine. Then she discontinued sibutramine, and hypomanic symptoms disappeared in a day, returning to her baseline atypical depression. She had never had hypomanic spontaneous episodes, and she had never had substance-related disorders. She had a first-degree relative with bipolar II disorder. The husband confirmed the information.

The close temporal relationship between onset and disappearance of hypomania with the start and discontinuation of sibutramine suggests a causal link. Hypomania is not listed among the adverse events of sibutramine in nonpsychiatric patients.[1] The timing of hypomania (rapid onset after sibutramine intake, symptoms closely associated with presence of the drug) suggests an effect like that of the dopamine-norepinephrine releasing agent *d*-amphetamine.[3,4] Sibutramine does not have amphetamine-like effects in stimulant users,[5,6] but some animal studies showed that it may increase extracellular brain dopamine levels as great as the levels of brain serotonin.[7] A case of the appearance of acute psychosis during sibutramine use may support these animal studies.[8] The rapid disappearance of hypomania after sibutramine discontinuation was probably related to its short half-life (less than 24 hours, including active metabolites).[9] When combined with a serotonin reuptake inhibitor (citalopram), sibutramine (an SNRI) might induce an amphetamine-like hypomania. A serotonin syndrome (believed to be caused by too much brain serotonin), related to combination of sibutramine-induced and citalopram-induced serotonin increase, might also have been the cause of hypomania (hypomania is among the symptoms of the serotonin syndrome).[10] However, this patient did not have hypomania when treated for some months with a high-dose combination of serotonin reuptake inhibitors (citalopram, 40 mg daily, plus fluoxetine, 60 mg daily), which should have greatly increased brain serotonin. However, the serotonin overload that occurred with sibutramine-citalopram may not be equivalent to the serotonin overload caused by citalopram-fluoxetine. Of the other symptoms of the serotonin syndrome (myoclonus, diaphoresis, shivering, tremor, diarrhea, incoordination, fever, and confusion),[9] only diaphoresis and shivering were present. This patient had elements of both a stimulant-related state and a serotonin syndrome. A positive family history for bipolar II disorder may have increased the vulnerability to the hypomania-inducing effects of the citalopram-sibutramine combination. She had never taken psychostimulants in the past. Whichever the mechanism, the report suggests that combining sibutramine and a serotonin reuptake inhibitor could induce hypomania in persons with unipolar major depressive disorder with bipolar family history.

References

1. James WPT, Astrup A, Finer N, et al, for the STORM Study Group. Effect of sibutramine on weight maintenance after weight loss: a randomized trial. Lancet 2000;356:2119–2125
2. First MB, Spitzer RL, Gibbon M, et al. Structured Clinical Interview for DSM-IV Axis I Disorders-Clinician Version (SCID-CV). Washington, DC: American Psychiatric Press; 1997
3. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition. Washington, DC: American Psychiatric Association; 1994
4. Heal DJ, Aspley S, Prow MR, et al. Sibutramine: a novel anti-obesity drug: a review of the pharmacological evidence to differentiate it from *d*-amphetamine and *d*-fenfluramine. Int J Obes Relat Metab Disord 1998;22(suppl 1):S18–S28
5. Cole JO, Levin A, Beake B, et al. Sibutramine: a new weight loss agent without evidence of the abuse potential associated with amphetamines. J Clin Psychopharmacol 1998;18:231–236
6. Schuh LM, Schuster CR, Hopper JA, et al. Abuse liability assessment of sibutramine, a novel weight control agent. Psychopharmacology (Berl) 2000;147:339–346
7. Balcioglu A, Wurtman RJ. Sibutramine, a serotonin reuptake inhibitor, increases dopamine concentrations in rat striatal and hypothalamic extracellular fluid. Neuropharmacology 2000;39: 2352–2359
8. Taflinski T, Chojnacka J. Sibutramine-associated psychotic episode. Am J Psychiatry 2000;157:2057–2058
9. Stock MJ. Sibutramine: a review of the pharmacology of a novel anti-obesity agent. Int J Obes Relat Metab Disord 1997; 21(suppl 1):S25–S29
10. Sternbach H. The serotonin syndrome. Am J Psychiatry 1991;148: 705–713

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Risperidone-Induced Pisa Syndrome

Sir: The Pisa syndrome is a rare adverse effect of neuroleptic medication that is characterized by a peculiar posture featuring lateral flexion and backward axial rotation of the trunk.[1] We report on a patient in whom this syndrome developed after 15 months of treatment with risperidone, which is one of the atypical neuroleptics with a high selectivity for dopamine D_2 receptors and serotonin 5-HT_2 receptors. As far as we know, this is the first report of risperidone-induced Pisa syndrome.

Case report. Mr. A was a 25-year-old man with a 5-year history of schizophrenia (DSM-IV 295.10). He had no family history of dystonia or other movement disorders and no abnormal findings on hematology tests, biochemistry tests, magnetic resonance imaging of the brain, and electroencephalography.

Because of severe auditory hallucinations, haloperidol (maximum dose = 8 mg/day) plus biperiden (maximum dose = 6 mg/day) was administered for 33 months after his first schizophrenic episode. However, risperidone (maximum dose = 9 mg/day) plus biperiden (maximum dose = 6 mg/day) was substituted after severe finger tremor developed.

Fifteen months after starting risperidone therapy (current dose = 7 mg/day), Mr. A complained of leaning to the right side and being unable to straighten up. Physical examination revealed tonic flexion of the trunk to the right accompanied by slight backward axial rotation. After withdrawal of risperidone, he was changed to haloperidol (3 mg/day) plus biperiden (3 mg/day), but his symptoms showed gradual progression. At 5 months after the onset of these symptoms, he was admitted to our psychiatric ward and was treated with haloperidol (3 mg/day) plus biperiden (3 mg/day) for psychosis. He also received agents reported to improve tardive dystonia, such as tiapride (maximum dose = 225 mg/day) for 4 weeks and clonazepam (maximum dose = 6 mg/day) for 7 weeks, but no benefit was seen. However, after 3 weeks of treatment with L-dopa/benserazide (400/100 mg/day), his symptoms improved slightly. Subsequently, 2 weeks after the addition of cabergoline (0.75 mg/day), the symptoms showed partial remission without exacerbation of his psychosis. Despite continued administration of these drugs, he still has these symptoms, although they are much improved.

Our patient fulfilled the criteria for tardive dystonia proposed by Burke et al.[2] He also had all the major features of the Pisa syndrome, so it appears that this syndrome developed as a form of tardive dystonia. Since atypical neuroleptics, including risperidone, have generally been considered to be less likely to cause tardive dystonia than typical neuroleptics, risperidone has been regarded as a useful therapeutic agent for patients with such dystonia.[3] Recently, it was reported that the Pisa syndrome could be caused by atypical neuroleptics like clozapine[4] and sertindole[5] in elderly patients with cerebral atrophy. Because our patient was a young man without cerebral damage, we conclude that the new neuroleptics can induce the Pisa syndrome in both elderly patients with brain damage and younger patients without it and that it may be very difficult to achieve complete remission despite various treatments. Therefore, the new neuroleptics should be administered with the same caution as classic agents.

REFERENCES

1. Ekbom K, Lindholm H, Ljungberg L. New dystonic syndrome associated with butyrophenone therapy. Z Neurol 1972;202:94–103
2. Burke RE, Fahn S, Jankovic J, et al. Tardive dystonia: late-onset and persistent dystonia caused by antipsychotic drugs. Neurology 1982; 32:1335–1346
3. Chong S-A, Remington G, Tan C-N. Risperidone treatment of tardive dyskinesia and dystonia [letter]. J Clin Psychiatry 1999;60:340–341
4. Kurtz G, Kapfhammer H-P, Peuker B. Pisa-Syndrom unter Clozapintherapie. Nervenarzt 1993;64:742–746
5. Padberg F, Stuebner S, Buch K, et al. Pisa syndrome during treatment with sertindole [letter]. Br J Psychiatry 1998;173:351–352

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Interferon-Induced Depression Treated With Citalopram

Sir: Chronic infection with hepatitis.65 C virus (HCV) is the most common cause of liver disease in the United States and is responsible for approximately one third of liver transplants.[1] Interferon-alpha and combination therapy with interferon-alpha and ribavirin are the only approved treatments for chronic hepatitis C[2]; however, side effects, which include depression, anxiety, irritability, and even suicidal thoughts,[3–8] can lead to discontinuation of therapy.

In the following case, a patient who had recovered from an initial depressive episode suffered a relapse following her diagnosis of HCV and initiation of interferon-alpha and ribavirin therapy. Citalopram, 40 mg/day, provided rapid, effective, and lasting relief from depressive symptoms and was well tolerated.

Case report. Ms. A, a 49-year-old menopausal woman, met DSM-IV criteria for major depression involving crying spells, low energy, anhedonia, insomnia, anxiety, and irritability. She reported no prior psychiatric history, she was taking no medications, and no comorbid conditions were evident at this time. Family history was significant for an 18-year-old daughter who had been diagnosed with cyclothymia.

Bupropion was prescribed, and the dose was escalated to 400 mg/day in 6 days' time. After 6 weeks of therapy with no benefit noted, but side effects of insomnia and anxiety reported, bupropion dosage was decreased to 300 mg/day and fluoxetine, 10 mg/day, and zolpidem, 10 mg at bedtime, were added. Four weeks after these adjustments, Ms. A had discontinued both antidepressants due to anorgasmia and decreased libido and remained on zolpidem at bedtime. At this visit, her Hamilton Rating Scale for Depression (HAM-D) score was 34, and citalopram, 20 mg/day, was prescribed. After 4 weeks of citalopram therapy, her HAM-D score was 12, and no sexual side effects were present. She reported only minor fatigue that had not resolved with the rest of her depressive symptoms. She was advised to remain on this medication for 6 months, and zolpidem was continued on an as-needed basis.

A routine physical examination and blood work 4 months after initiation of citalopram therapy revealed the presence of HCV, and transaminase levels were elevated (AST = 110 IU/L; ALT = 174 IU/L), while thyrotropin and B_{12} levels were normal (1.32 μIU/mL and 360 pg/mL).

Within 48 hours of commencing combination therapy with interferon-alpha (3 million units subcutaneously 3 times

a week) and ribavirin (400 mg in the morning and 600 mg in the evening), Ms. A experienced crying spells, anhedonia, and anorexia and found it difficult to get out of bed in the morning. She reported that this depressive episode was more severe than the previous one described (confirmed by a HAM-D score of 36). Citalopram dosage was increased to 40 mg/day and zolpidem was continued as needed (10 mg at bedtime). Within 2 weeks, her depressive symptoms resolved and her HAM-D score returned to a baseline level. She remained on citalopram therapy through the 6 months of interferon-ribavirin therapy without recurrence of the depressive episode. ALT, AST, B_{12}, and thyrotropin levels were all normal at the conclusion of therapy, and alkaline phosphatase levels remained normal throughout her illness and treatment.

Two months after completion of treatment for HCV, citalopram was tapered and discontinued over a 2-week period; zolpidem was being used about 10 days per month at this time. If further therapy for HCV should be needed, Ms. A has consented to beginning prophylactic antidepressant therapy prior to HCV treatment.

Interferon-alpha therapy is known to carry the risk of psychiatric symptoms in patients who are otherwise euthymic and can trigger a recurrence of depression in patients who had responded to antidepressant therapy.[9] Furthermore, there appears to be an increased prevalence of depression in patients with chronic HCV infection.[10] In the above case, the patient responded favorably and quickly to an increase in citalopram dose to 40 mg/day despite concurrent hepatic disease and treatment, which is consistent with reports of successful treatment of interferon-induced mood disorders with antidepressants.[7,11,12] It has been suggested that the serotonergic system is possibly influenced by interferon,[13,14] and this patient has consented to initiation of antidepressant therapy prior to any further HCV therapy in the future. Indeed, a recent study[15] argues for just such prophylactic antidepressant therapy in interferon-alpha–treated patients. In this double-blind study, malignant melanoma patients undergoing interferon-alpha treatment were much less likely to suffer from depression, and thus less likely to drop out of therapy, when receiving paroxetine, than those patients taking placebo from the outset of HCV treatment.[15]

Dr. Farah has been a consultant for GlaxoSmithKline; has received honoraria from Forest, GlaxoSmithKline, Pfizer, and Lilly; and is a speaker/advisory board member for Forest, Lilly, GlaxoSmithKline, Pfizer, and Cephalon.

References

1. Interferon plus ribavirin for chronic hepatitis C. Med Lett Drugs Ther 1999;42:53–54
2. Gleason OC, Yates WR. Five cases of interferon alpha-induced depression treated with antidepressant therapy. Psychosomatics 1999;40:510–512
3. Janssen H, Brouwer J, van der Mast R, et al. Suicide associated with alpha-interferon therapy for chronic viral hepatitis. J Hepatol 1994;21:241–243
4. Okanoue T, Sakamoto S, Itoh Y, et al. Side effects of high-dose interferon therapy for chronic hepatitis C. J Hepatol 1996;25: 283–291
5. Renault F, Hoofnagle JH, Park Y, et al. Psychiatric complications of long-term interferon-alpha therapy. Arch Intern Med 1987;147: 1577–1580
6. Sorrell MF. A 47-year-old man with asymptomatic hepatitis C infection. JAMA 1996;275:1023–1029
7. Yokoyama A, Kimura Y, Shigemura J. Psychiatric side effects of interferon. J Toxicol Sci 1996;21:93–96
8. Dieperink E, Willenbring M, Ho SB. Neuropsychiatric symptoms associated with hepatitis C and interferon alpha: a review. Am J Psychiatry 2000;157:867–876
9. McAllister-Williams RH, Young AH, Menkes DB. Antidepressant response reversed by interferon. Br J Psychiatry 2000;176:93
10. Lee DH, Jamal H, Regenstein F, et al. Morbidity of chronic hepatitis C as seen in tertiary-care medical center. Dig Dis Sci 1997;42: 186–191
11. Levenson JL, Fallon HJ. Fluoxetine treatment of depression caused by interferon-alpha. Am J Gastroenterol 1993;88:760–761
12. Goldman LS. Successful treatment of interferon alpha-induced mood disorder with nortriptyline. Psychosomatics 1994;35:412–413
13. Werner-Felmayer G, Werner ER, Fuchs D, et al. Characteristics of interferon induced tryptophan metabolism in human cells in vitro. Biochim Biophys Acta 1989;1012:140–147
14. Delgado PL, Price LH, Miller HL, et al. Rapid serotonin depletion as a provocative challenge test for patients with major depression: relevance to antidepressant action and the neurobiology of depression. Psychopharmacol Bull 1991;27:321–330
15. Dominique L, Musselman DH, Lawson JF, et al. Paroxetine for the prevention of depression induced by high-dose interferon alfa. N Engl J Med 2001;344:961–966

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Clozapine and Tardive Dyskinesia

Sir: In general, it is assumed that clozapine does not cause tardive dyskinesia. In fact, clozapine is used to treat the often permanent and disabling condition.[1] A few reports, however, have demonstrated that tardive dyskinesia, although rare, can result from clozapine use.

Doepp and Buddeberg[2] described a patient with chronic perioral dyskinesia and akathisia who developed new-onset choreoathetoid dyskinesia of the trunk, extremities, and face after 23 days of treatment that quickly resolved with clozapine discontinuation. Additionally, a case of jaw dyskinesia was reported, occurring 2 weeks after clozapine was introduced.[3] The symptom persisted for more than 1 year while the patient was treated with clozapine. Also, Dave[4] described 2 patients who appeared to have developed clozapine-induced tardive dyskinesia. In the largest study of clozapine-related tardive dyskinesia to date, Kane and colleagues[5] studied 28 patients treated with clozapine for at least 1 year, with an average of 7.7 years. Results were inconclusive, as 2 patients in the study were diagnosed with tardive dyskinesia at the conclusion but had questionable tardive dyskinesia at baseline. We report a case of a patient who developed symptoms of tardive dyskinesia while treated with clozapine.

Case report. Ms. A, a 45-year-old woman with DSM-IV schizoaffective disorder, bipolar type, had been treated with haloperidol in the past; however, regular use of this medication was discontinued 3 years earlier. Ms. A underwent trials of risperidone and olanzapine. Neither was effective in reducing auditory hallucinations or establishing mood stabilization.

Ms. A had never experienced symptoms of tardive dyskinesia prior to commencing treatment with clozapine. She first experienced involuntary tongue movements and akathisia at a clozapine dosage level of 225 mg/day, 5 months after clozapine treatment was initiated. She was hesitant to decrease the dose, because clozapine was the only effective medication for treating her hallucinations. Symptoms of tardive dyskinesia resolved when the dose was decreased to 150 mg/day; however, Ms. A experienced a distressing increase in auditory hallucinations.

The dose of clozapine was then gradually increased to 250 mg/day. Although it resulted in a decrease in auditory hallucinations, the involuntary tongue movements returned. Because clozapine had been so effective in decreasing her auditory hallucinations, Ms. A decided to accept the risk of continued tardive dyskinesia.

This case demonstrates that clozapine may cause tardive dyskinesia. Like most patients on clozapine therapy, Ms. A had been exposed to other antipsychotics, which may have sensitized her to develop tardive dyskinesia. Animal models have also demonstrated that clozapine can cause involuntary movements, although at lower rates than with haloperidol.[6] Thus, because tardive dyskinesia is a serious adverse reaction, patients treated even with clozapine should be monitored for its development.

The authors report no financial or other relationships related to the subject matter in this communication.

References

1. Spivak B, Mester R, Abesgaus J, et al. Clozapine treatment for neuroleptic-induced tardive dyskinesia, parkinsonism, and chronic akathisia in schizophrenic patients. J Clin Psychiatry 1997;58: 318–322
2. Doepp S, Buddeberg C. Extrapyramidale symptome unter clozapin. Nervenarzt 1975;46:589–590
3. de Leon J, Moral L, Camunas C. Clozapine and jaw dyskinesia: a case report. J Clin Psychiatry 1991;52:494–495
4. Dave M. Clozapine-related tardive dyskinesia. Biol Psychiatry 1994;35:886–887
5. Kane JM, Woerner MG, Pollack S, et al. Does clozapine cause tardive dyskinesia? J Clin Psychiatry 1993;54:327–330
6. Tamminga C, Thaker G, Moran M, et al. Clozapine in tardive dyskinesia: observations from human and animal model studies. J Clin Psychiatry 1994;55(9, suppl B):102–106

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Role of Receptor Binding Profiles in Antidepressant-Induced Sexual Dysfunction

Sir: Montejo et al.[1] studied the incidence of sexual dysfunction associated with antidepressant agents. One thousand twenty-two subjects treated with SSRIs, venlafaxine, mirtazapine, nefazodone, moclobemide, or amineptine were interviewed when they visited an outpatient clinic. The authors not only reported very interesting data about the incidence of sexual dysfunction, but they also measured the incidence of spontaneous improvement of sexual dysfunction. Among 352 patients, 55 patients (15.6%) reported "spontaneous improvement sometime after the onset of treatment." The authors conclude that "it does not seem to be practical" to wait for tolerance to develop. I agree with the authors that, until now, specific data on the mean time required for sexual side effects to disappear and on the probability of spontaneous remission were missing. But I don't think that the data presented by Montejo et al. are specific enough to draw conclusions for the management of antidepressant-induced sexual dysfunction, because they included patients treated with various antidepressants that had different receptor binding profiles. Compared with tricyclic antidepressants, SSRIs have a more specific receptor binding profile, as well as a specific sexual side effect profile. Although decreased sexual desire and arousal (erection, lubrication) difficulties have been associated with SSRI treatment, delayed or absent orgasm seems to be the typical sexual dysfunction caused by SSRIs. Venlafaxine, mirtazapine, nefazodone, moclobemide, and amineptine might cause different kinds of sexual dysfunction (libido, arousal, orgasmic function) with different outcome.

Reference

1. Montejo AL, Llorca G, Izquierdo JA, et al, for the Spanish Working Group for the Study of Psychotropic-Related Sexual Dysfunction. Incidence of sexual dysfunction associated with antidepressant agents: a prospective multicenter study of 1022 outpatients. J Clin Psychiatry 2001;62(suppl 3):10–21

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Dr. Montejo Replies

Sir: I agree with Dr. Haberfellner that the results reported by my coworkers and I[1] about spontaneous remission of sexual dysfunction are not specific enough regarding each drug. We must wait to obtain new results comparing spontaneous remission of antidepressants with different receptor binding profiles. My colleagues and I are working on increasing the sample in order to have data about this relevant issue. Nevertheless, serotonergic drugs seem to have very consistent results related to the incidence of libido, orgasm, ejaculation, and also erectile dysfunction, not only with selective serotonin reuptake inhibitors (SSRIs) but also with clomipramine and venlafaxine. In our study, patients on venlafaxine treatment (N = 55, mean dosage = 159 mg/day) showed incidence rates of sexual dysfunction similar to those of patients on SSRI treatment. This could be due to the serotonergic mechanism of action of venlafaxine (decreased libido, 60.0%; delayed orgasm/ejaculation, 61.9%; anorgasmia/no ejaculation, 41.8%; and erectile dysfunction, 40.0%). These data did not show statistical differences with SSRIs. On the other hand, mirtazapine and particularly nefazodone and moclobemide are associated with significantly less incidence of sexual dysfunction than are SSRIs and venlafaxine. These different rates of incidence of libido and orgasmic problems could be due to a different mechanism of action. Unfortunately, amineptine was removed from the market last year in Europe, so we can no longer use it as an alternative therapy. It would be informative to analyze different outcomes related to each drug and be able to predict whether some of them could have a higher rate of spontaneous remission of sexual dysfunction than others. I would like to thank Dr. Haberfellner for his suggestive and thought-provoking comments.

Reference

1. Montejo AL, Llorca G, Izquierdo JA, et al, for the Spanish Working Group for the Study of Psychotropic-Related Sexual Dysfunction. Incidence of sexual dysfunction associated with antidepressant agents: a prospective multicenter study of 1022 outpatients. J Clin Psychiatry 2001;62(suppl 3):10–21

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Olanzapine for the Treatment of Monosymptomatic Hypochondriacal Psychosis

Sir: The syndrome of monosymptomatic hypochondriacal psychosis is a form of DSM-IV delusional disorder, somatic subtype, characterized by a delusional belief that one is afflicted with a medical disorder or defect. The delusional symptoms are circumscribed. Such patients often present to dermatologists with delusions of parasitosis. The literature describes case studies and 1 double-blind crossover study indicating benefit from typical neuroleptics, especially pimozide, for this disorder.[1–3] However, pimozide has the potential undesirable effects of typical neuroleptics, including tardive dyskinesia and other extrapyramidal symptoms, and significant cardiac conduction effects, such that the manufacturer recommends baseline and follow-up electrocardiograms with pimozide treatment. Several case studies describe successful treatment of monosymptomatic hypochondriacal psychosis with atypical neuroleptics, 3 with risperidone,[4–6] 1 with sertindole,[7] and 1 with olanzapine.[8] The present case describes a second successful treatment of monosymptomatic hypochondriacal psychosis with the atypical antipsychotic olanzapine.

Case report. Ms. A, a 76-year-old woman, was referred for psychiatric treatment by her internist for persisting delusions of about 5 years' duration that her body was infected by fungus. She said the fungus caused a funny taste in her mouth, and, when she washed dishes, she could see the fungus coming out of her skin and going into the water. She thought the fungus crept down her arms during the night. She spent about an hour in the bathtub in the morning and again in the evening, scrubbing her skin with a washcloth to remove the fungus. She avoided some social situations to avoid contaminating other people. Her appetite was poor, and her weight had decreased from approximately 50 kg (110 lb) to about 37 kg (82 lb) over several years. She slept adequately and described her mood as neutral; she scored only 4 on the Beck Depression Inventory in the normal range. She had a brief unsuccessful trial of pimozide at unknown dosage from a dermatologist. Lorazepam had provided no benefit. Ms. A also had a mild dementia of the Alzheimer type, which, according to her family and to her neurologist's records, became manifest years after the somatic delusional disorder. Her dementia was treated with donepezil, 10 mg/day. She had also been diagnosed with Parkinson's disease several years after the onset of delusions and started on levodopa/carbidopa, 25/100 mg t.i.d. She took levothyroxine, 0.075 mg/day, for hypothyroidism. On mental status examination, the patient had a blunted affect and subdued mood and no other hallucinations or delusions, save those involving her nonexistent fungal infection. She was disoriented to time, with poor short-term memory, scoring 18 of 30 on the Mini-Mental State Examination.

Treatment with olanzapine at 2.5 mg/day improved her delusions in a matter of weeks. Ms. A reported that her skin was doing well, and the family noted that she was no longer preoccupied with the fungus. She stopped her excessive bathing, her affect appeared brighter, and neither her mild Parkinson's disease nor her dementia appeared to worsen on the low dose of olanzapine. After 5 months of treatment, Ms. A gained 6 kg of weight (13 lb), although she still subjectively reported no appetite. Her remission continues after 9 months. Although the olanzapine treatment has not been interrupted to see if delusions recur, the dramatic impact on her long-term delusions within weeks of starting olanzapine suggests a real pharmacologic effect.

Olanzapine may be another agent, along with risperidone, to consider as first-line treatment of monosymptomatic hypochondriacal psychosis because it has a more benign side effect profile than pimozide or other typical neuroleptics.

Financial disclosure: Dr. Fawcett has served on the speakers board for Janssen.

References

1. Munro A. Monosymptomatic hypochondriacal psychosis. Br J Psychiatry 1988;153(suppl 2):32–36
2. Koo J, Gambla C. Delusions of parasitosis and other forms of monosymptomatic hypochondriacal psychosis. Dermatol Clin 1996;14:429–438
3. Baker BB, Cook BL, Winokur G. Delusional infestation: the interface of delusions and hallucinations. Psychiatr Clin North Am 1995;18: 345–361
4. Cetin M, Ebrinc S, Agargun NY, et al. Risperidone for treatment of monosymptomatic hypochondriacal psychosis [letter]. J Clin Psychiatry 1999;60:554
5. Galluci G, Beard G. Risperidone and the treatment of delusions of parasitosis in an elderly patient. Psychosomatics 1995;36:578–580
6. Safer DL, Wenegrat B, Roth WT. Risperidone in the treatment of delusional parasitosis: a case report. J Clin Psychopharmacol 1997;17:131–132
7. Yorston G. Treatment of delusional parasitosis with sertindole. J Geriatr Psychiatry 1997;12:1127–1128
8. Weintraub E, Robinson C. A case of monosymptomatic hypochondriacal psychosis treated with olanzapine. Ann Clin Psychiatry 2000;1:247–249

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Retraction

Sir: In our article "Safety and Tolerability of a Rapidly Escalating Dose-Loading Regimen for Risperidone" in the December 2000 issue (J Clin Psychiatry 2000;61:909–911), we reported safety and tolerability of a rapid-loading regimen employed clinically on our inpatient service for a period of time. The safety and tolerability data were collected by means of a retrospective chart review. Unfortunately, the article did not describe this methodology explicitly. In order to avoid potential confusion by readers, we ask that this article be retracted from the scientific literature. We also request formally that the National Library of Medicine annotate the MEDLINE database with the retraction.

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