had been warned to expect headaches, these being among the common side effects of SSRIs.

Breslau and associates[5] investigated the association between migraine and major depression. They noted the usefulness of tricyclic antidepressants in both conditions and the possible role of serotonin. Studying a sample of 1007 young adults, they found that migraine predisposed to depression, and depression to migraine, and proposed that a shared mechanism explained this bidirectional comorbidity. It may be that the more effective the antidepressant, the more likely it will alleviate migraines.

References

1. Adly C, Straumanis J, Chesson A. Fluoxetine prophylaxis of migraine. Headache 1992;32:101–104
2. Saper J. Double-blind trial of fluoxetine: chronic daily headache and migraine. Headache 1994;34:497–502
3. Bank J. A comparative study of amitriptyline and fluvoxamine in migraine prophylaxis. Headache 1994;34:476–478
4. Foster CA, Bafaloukos J. Paroxetine in the treatment of chronic daily headache. Headache 1994;34:587–589
5. Breslau N, Davis GC, Schultz LR, et al. Migraine and major depression: a longitudinal study. Headache 1994;34:387–393

Peter Hays, M.B., B.S., F.R.C.P.C.
Alberta, Canada

An Open-Label Study of SSRI Treatment in Depressed Hispanic and Non-Hispanic Women

Sir: Symptoms of depression are common across cultures, but the clinical presentation of depression may differ from country to country and, within the same country, differ between ethnic groups.[1,2] Among Hispanic patients, somatization is a more common presentation of depression than among non-Hispanic patients with major depression, and an increased prevalence of somatic symptoms in Hispanic patients with major depression suggests that they present more frequently at primary care rather than psychiatric facilities.[3–7] It is reported that Hispanic subjects may be more sensitive to the somatic side effects of pharmacotherapy.[8] Marcos and Cancro[9] found that depressed Hispanic women received lower doses of tricyclic antidepressants and had more problems with side effects than depressed non-Hispanic patients.

Twenty-six female patients (13 Hispanic and 13 non-Hispanic) were enrolled in an open-label study of paroxetine (N = 18) and fluoxetine (N = 8) at the Aurora Hispanic Center and the Mental Health Clinical Research Center, University of California, San Diego. Subjects were medically healthy and had DSM-III-R diagnosis of major depression.[10] The Hamilton Rating Scale for Depression (HAM-D)[11] and a 17-item somatic checklist were administered weekly. Data were checked for normal distribution and homogeneity of variance; analysis of variance (ANOVA), t tests, chi-square tests, and the Fisher's exact test were performed.

The mean ± SD age of the Hispanic group was 33.0 ± 14.0 years versus 44.8 ± 12.1 for the non-Hispanic group (t = 2.3, df = 24, p = .03). The mean ± SD years of education for the Hispanic group was 8.7 ± 2.8 years versus 14.9 ± 2.6 for the non-Hispanic group (t = 5.9, df = 24, p = .0004). One Hispanic patient reported a previous episode of major depression; 9 non-Hispanic patients reported previous episodes (χ^2 = 11.5, df = 1, p = .0007). Four subjects in each group presented with lifetime comorbid DSM-III-R Axis I diagnoses (diagnoses similar for both groups). The initial HAM-D score was 19.0 ± 3.8 for the Hispanic group and 19.5 ± 4.7 for the non-Hispanic group (t = .32, df = 24, p = .7). The final HAM-D scores were 6.6 ± 6.8 for the Hispanic group and 5.3 ± 7.8 for the non-Hispanic (F = .70, df = 1,15; p = .415). When age was covaried, the result did not change.

The groups were identical in type and number of somatic symptoms endorsed at baseline. Six Hispanic and 3 non-Hispanic subjects terminated early (χ^2 = 1.5, df = 1, p = .216); reasons were identical for both groups: noncompliance, intolerable side effects, and pregnancy. Hispanic subjects averaged 2.2 ± 2.0 side effect complaints versus 5.1 ± 2.5 for non-Hispanic subjects (t = 3.18, df = 24, p < .005). There were no differences in response, side effects, or attrition rates between fluoxetine- and paroxetine-treated subjects.

Ours is the first prospective study to compare response rates, side effect profiles, and attrition rates for Hispanic and non-Hispanic patients from the same area who were treated with serotonin selective reuptake inhibitors. We believe a larger masked prospective study is warranted comparing treatment of depressed patients from different ethnic groups.

The study was funded by SmithKline Beecham Pharmaceuticals.

References

1. Kleinman A. Somatization and the "new cross-cultural psychiatry." Soc Sci Med 1977;11:3–10
2. Singer K. Depressive disorders from a transcultural perspective. Soc Sci Med 1975;9:289–301
3. Angel R, Guarnaccia P. Mind, body, and culture: somatization among Hispanics. Soc Sci Med 1989;28:1229–1238
4. Kolody B, Vega W, Meinhardt K, et al. The correspondence of health complaints and depressive symptoms among Anglos and Mexican-Americans. J Nerv Ment Dis 1986;174(4):221–228
5. Mezzich J, Raab E. Depressive symptomatology across the Americas. Arch Gen Psychiatry 1980;37:818–823
6. Escobar J, Gomez J, Tuason V. Depressive phenomenology in North and South American patients. Am J Psychiatry 1983;140:47–51
7. Escobar J, Karno M, Golding J, et al. Psychosocial influences on psychiatric symptoms: the case of somatization. In: Gaviria M, Arana J, eds. Health and Behavior: Research Agenda for Hispanics (Simon Bolivar Research Monograph Series). Chicago, Ill: The University of Illinois at Chicago; 1987:207–215
8. Escobar J, Tuason V. Antidepressant agents: a cross cultural study. Psychopharmacol Bull 1980;16(3):49–52
9. Marcos L, Cancro R. Pharmacotherapy of Hispanic depressed patients: clinical observations. Am J Psychother 1982;36(4):505–512
10. Spitzer R, Williams J, Gibbon M, et al. Instruction manual for the Structured Clinical Interview for DSM-III-R (SCID). New York, NY: New York State Psychiatric Institute; 1988
11. Hamilton M. A rating scale for depression. J Neurol Neurosurg Psychiatry 1960;23:56–62

Margarita Alonso, M.D.
Eduardo Val, M.D.
Mark Hyman Rapaport, M.D.
San Diego, California

Akathisia as Violence

Sir: Manifestations of akathisia, a side effect of antipsychotic drugs, include restlessness, muscular tension, and a compulsion to move.[1] Infrequently, agitation and violence have been reported to be associated with antipsychotic treatment and could be related to akathisia.[2,3] Differentiating between akathisia that manifests itself as violence and generalized psychotic agitation is clinically important in order to avoid a vicious circle of violence in patients who are being treated with antipsychotics. We report a case of persistent agitation and violence in a patient with bipolar mood disorder that was probably a manifestation of akathisia.

Case Report. Mr. A, a 47-year-old white man with a diagnosis of bipolar mood disorder, was brought to the emergency room because he was screaming in the streets. Mr. A had over 30 past psychiatric admissions associated with agitation and violence and was often discharged against medical advice. He was nearly always noncompliant with his antipsychotic medications, claiming that they made him "jump and lose my temper." Prior to the present admission, Mr. A's daily medications included haloperidol 20 mg, lithium carbonate 1500 mg, divalproex sodium 500 mg, and benztropine 1 mg. At admission, the patient was grandiose, had loud and pressured speech, and admitted he was not taking haloperidol. He was given haloperidol 15 mg q.h.s. and benztropine 1 mg q.a.m. Within 24 hours he started pacing; became restless, agitated, and violent; complained of feeling "jumpy"; and attacked a staff member. On Day 5 of his hospitalization, haloperidol and benztropine were discontinued; chlorpromazine was started, and the dose was increased to 950 mg/day. Mr. A, although sedated, remained threatening and violent. On Day 13, chlorpromazine was discontinued, and haloperidol was restarted at a higher dose of 15 mg p.o. b.i.d. Mr. A again complained of "jumpiness" and punched a television cabinet, causing a self-inflicted fracture. On hospital Day 17, owing to an error, haloperidol was discontinued. The patient became calmer, less irritable, displayed no angry outbursts, and required no further room restrictions. After 5 days, when the error was discovered, haloperidol was restarted at a lower daily dose of 10 mg. Within 3 days, the patient became violent and required room restriction. Haloperidol was then discontinued, the patient's agitation and violence resolved, and a week later he was discharged. His daily medications were lithium carbonate 1500 mg (serum level = 0.9 mEq/L; this dose had not been changed during his hospitalization), lorazepam 1 mg, and divalproex sodium 500 mg. On these medications, he remained well 6 months postdischarge, his longest period as an outpatient.

The association between antipsychotic administration, akathisia, and violence in psychiatric patients has been noted in two reports.[2,3] Herrera et al.[2] showed a trend for more violent episodes to occur with haloperidol than with placebo or low-potency neuroleptics. Crowner et al.[3] found that for violent psychiatric patients taking antipsychotics, half of the assailants had akathisia before the assaults, while only 20% of nonviolent patients had akathisia. However, to support a causal relationship between antipsychotic administration, akathisia, and violence, it is necessary to document a clear onset of akathisia and violent behavior upon initiation of antipsychotic treatment and resolution of both with antipsychotic discontinuation. Although agitation and violence result from a severe manic episode, Mr. A's case documents such an association: on two occasions, the onset and the resolution of both his "jumpiness" and his violent behavior coincided with the beginning and the ending of antipsychotic medication treatment. The fact that the jumpiness occurred with haloperidol and not with chlorpromazine is another factor indicative that Mr. A has exhibited akathisia rather than nonspecific activation of mania; this is because akathisia is more common with higher potency as compared with low-potency neuroleptics. One can also speculate that Mr. A's rocky clinical history was related to aggressive behavior perpetuated by antipsychotic administration. The possibility that aggressive and violent behavior unresponsive to antipsychotic treatment could be a variant of akathisia should be included in the differential diagnosis of acute psychosis and in alternative treatment strategies for bipolar mood disorder. Benzodiazepines in combination with lower neuroleptic doses, lithium, or valproate should be considered.

References

1. Braude WM, Barnes TRE, Gore SM. Clinical characteristics of akathisia: a systematic investigation of acute psychiatric inpatient admissions. Br J Psychiatry 1983;143:139–150
2. Herrera JN, Sramek JJ, Roy S, et al. High potency neuroleptics and violence in schizophrenics. J Nerv Ment Dis 1988;176:558–561
3. Crowner ML, Douyon R, Convit A, et al. Akathisia and violence [letter]. Psychopharmacol Bull 1990;26(1):115

Igor I. Galynker, M.D., Ph.D.
Deborah Nazarian, M.D.
New York, New York

Cessation of Self-Mutilation in a Patient With Borderline Personality Disorder Treated With Naltrexone

Sir: Self-mutilation and self-injurious behavior may be due in part to dysregulation of endorphin neurotransmitter systems.[1–3] Self-mutilation and dissociation occur frequently in patients with borderline personality disorder and associated histories of traumatic abuse and neglect, suggesting there may be an etiologic relationship between trauma, neglect, endorphin system dysfunction, and self-mutilation.[4–9] This hypothesis is supported by the observation that some patients with borderline personality disorder have elevated pain thresholds, a phenomenon mediated in part by endorphin systems.[12] The interruption of the reinforcing effects of self-mutilation on endorphin systems by opioid receptor antagonists may help to reduce self-mutilation in selected patients with borderline personality disorder. Although the literature is conflicting, several studies from the developmental disabilities literature have in fact demonstrated a reduction or cessation of self-mutilation behaviors with opioid antagonists.[10–13] Their benefit for patients with severe trauma histories and borderline personality disorder remains unstudied, although others have speculated on their potential usefulness.[14] I now present a case in which naltrexone treatment resulted in a dramatic and near complete cessation of self-mutilation in a patient with borderline personality disorder.

Case report. Ms. A, a 28-year-old woman with a history of profound trauma and neglect, suffered from borderline personality disorder, recurrent severe major depression, dysthymic disorder, and alcohol dependence. She also experienced "bad" thoughts about herself that were obsessive and ego-dystonic in nature. I initially began working with her 3 years ago in conjunction with her individual psychotherapist to help reduce her depression and suicidality. She experienced strong urges to cut herself during times of interpersonal stress at work or in psychotherapy, and she cut her arms with a razor during these times to relieve these urges and reduce her tension. This behavior occurred as many as several times per week, was accompanied by frequent visits to the emergency room for sutures, and had persisted for almost a decade despite 3 years of psycho-