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In This Issue



We conclude the year 2012 at the *Residents' Journal* with a variety of topics. Editor-in-Chief Monifa Seawell, M.D., begins with an editorial on this year's recipient of the *Residents' Journal* Editor's Choice award. Nicole Zuber, M.D., discusses the cultural and bicultural challenges involved in treating adolescent immigrants at risk for suicidal behaviors. Dylan P. Murray, M.D., presents a case report of acute alcohol withdrawal syndrome. Chris A. Karampahtsis, M.D., M.P.H. presents a case of a young woman with food-triggered panic attacks. Danijela Ivelja-Hill, M.D., Eileen Zhivago, M.D., and Maria del Pilar Trelles-Thorne, M.D., discuss the importance of early recognition of neuroleptic malignant syndrome, particularly in vulnerable populations. Iman Parhami, M.D., M.P.H., provides a clinical review of hypersexual disorder. Last, Jonathan R. Scarff, M.D., discusses bethanechol as treatment for antidepressant-associated sexual dysfunction.

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2012 Residents' Journal Editor's Choice

Monifa Seawell, M.D.
Editor-in-Chief

As I reflect on the *Residents' Journal* over the past 12 months, I am extremely pleased by the progress the Journal has made and by the large number of high-quality manuscripts that the Journal has featured. Our authors have educated our readers about a wide variety of topics, and through their dedication and hard work, the *Residents' Journal* has had another successful year.

This year, the *Residents' Journal* editorial staff selected a single manuscript to receive the distinction of Editor's Choice. The Editor's Choice award is given to an author whose manuscript represents a significant contribution to the *Residents' Journal* and to our readership. We will continue this tradition hereafter.

Among the many fine manuscripts that we received over the past year, "Bath Salts": Emergence of an Epidemic, by George Loeffler, M.D. (1), particularly stood out. Dr. Loeffler's article was selected for the 2012 *Residents' Journal* Editor's Choice award because of its high educational value, pertinence, timeliness, and broaching of an important subject matter that can have serious implications for the patients we serve.

Dr. Loeffler's article was also formally recognized in the December 2012 issue of the *American Journal of Psychiatry* (AJP). My overview of his manuscript, as featured in AJP, can be accessed [online](#). This is also available in [Spanish](#). Dr. Loeff-

fler's article was published in the [March 2012](#) issue of the *Residents' Journal*.

Join me in congratulating Dr. George Loeffler, from the Department of Psychiatry at the Naval Medical Center in San Diego, on this tremendous accomplishment and on his outstanding contribution to the *Residents' Journal*.

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1. Loeffler G: "Bath salts:" emergence of an epidemic. *Res J Am J Psychiatry* 2012; 7:13-15

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Immigration and Mental Health in Adolescents

Nicole Zuber, M.D.

The United States population is rapidly growing and diversifying. As reported by the U.S. Census Bureau, the Hispanic/Latino American population is projected to increase to between 62 and 133 million by the year 2050 (1). Immigrating to the United States involves an assortment of challenges, dangers, and other issues. Studies have found that Latino youths experience more anxiety-related and delinquency behaviors, depression, and drug use than non-Hispanic Caucasian youths. Similarly, reports have shown that Hispanic youths have higher rates of suicidal ideation, plans, and gestures than their non-Hispanic peers (2, 3). The present article examines the case of a Hispanic adolescent female who struggled with mental health issues as she immigrated and adapted to American culture and society.

Clinical Vignette

“Marie” is an adolescent, Mexican-born female with no psychiatric history until 2 years ago, when she began cutting herself, developed anxiety symptoms, and had suicidal thoughts. She presented after attempting suicide by overdosing on numerous acetaminophen pills and cold medications. This was her second suicide attempt in 4 months. She currently lives in an urban U.S. city with her biological parents, who are illegal immigrants, and a younger brother, who is a U.S. citizen,

Marie lived between Mexico and the United States during the early years of her life. In Mexico, she was cared for by her maternal grandparents, along with her brother, while her parents remained in the United States in search of better financial and employment opportunities. When she was 5 years old, her brother was brought back to the United States, and she remained alone with her grandparents. She reports poor treatment by her grandparents while alone in Mexico. She

felt sad, angry, lonely, and disconnected from others, especially her parents. She was happiest when around her extended family but reports that resources were limited and that she had multiple chores.

She returned to the United States when she was 7 years old and reports that the journey was long, arduous, and difficult. She has done very well academically but has struggled with missing her extended family, learning English as a second language, figuring out the American culture, and connecting with her parents and friends.

She reports feeling increasingly depressed and anxious over the past 2 years because of increased academic demands after receiving a scholarship to attend a private school. She feels as though she is not able to “measure up” to the school and family pressures of success. In addition, she reports feeling like an outsider at school because peers have called her “odd and weird.” She has felt shameful for having attempted suicide in the past, has been concerned about her immigration status, and has felt disconnected from her parents’ cultural views: American versus Mexican.

Discussion

Migration involves changes that affect families collectively and individually. The experience can be associated with an array of issues, including leaving behind valued family members and friendships in the old country, learning a new language, trying to fit into a different sociocultural environment, adjusting to a new education system, and, at times, struggling against discrimination, prejudice, and feelings of isolation. Various groups are affected by the changes in different ways, and sometimes the changes can precipitate postmigration mental health issues. The cultural transition is seen by some as a psychological and sociological pro-

cess with significant implications for the mental health of immigrants (4).

Acculturation can be seen as a process in which cultural changes result from contact between two independent cultural groups, with the nondominant group being strongly influenced to take on the norms, values, and behaviors of the dominant group, with assimilation being the end point in the process (5). Biculturalism allows for the validation and reaffirmation of an individual’s identity by both cultures. It corroborates both traditional values and competencies in the “new” culture, allowing an individual to consolidate his or her sense of self (4).

Hispanic cultures are often embedded in a sense of “familism.” Familism is a perspective in which most activities are centered on the family unit, family loyalty, and family solidarity, with a high value placed on family cohesiveness, parental authority, and interdependence, and family goals often supersede individual ones. This sense of cohesiveness among Latino immigrants can be affected by the migration process, causing either comfort or distress (6, 7).

Researchers have shown that the effect of acculturation/assimilation into mainstream U.S. culture on Latino behaviors and health outcomes is very complex and not well understood, with both positive and negative behavioral outcomes. Studies have shown that among Latino youths, symptoms of depression have been associated with bicultural stress. Acculturation differences generate conflicts between adolescents and their parents, substance abuse issues, conduct problems, dating violence, and sexual behavior concerns (4–6).

As reported by the Centers for Disease Control and Prevention (CDC), suicide is the third leading cause of death for youths between the ages of 10 and 24 years. Cul-

tural variations in suicide rates exist, with Native American/Alaskan Native and Hispanic youths having the highest rates of suicide-related fatalities. The CDC also reported that Hispanic youths were more likely to report attempting suicide than their non-Hispanic peers (2). Hispanic adolescent females struggle with many complicated forces, including normal developmental processes, social and peer group factors, cultural traditions and bicultural challenges, unique individual characteristics, and family dynamics and relations. One study showed that among Latina daughters of immigrants with a low level of acculturation, suicidal acts commonly occur when these daughters cannot reconcile their acculturation differences with their parents (3). Another study showed that Latinas report high rates of hopelessness, suicide plans, and suicide attempts (8).

Various psychological and psychiatric evaluations were conducted with the adolescent in the above clinical vignette, highlighting her mood symptoms of anxiety and depression, struggles with the growing cultural divide between herself and her parents, and future concerns as related to her immigration status. She was started on an antidepressant to target her anxiety and depressive symptoms. A focus of treatment was on biweekly individual and weekly family therapy to help with these dynamic issues individually and collectively.

Assessment

Various tools can be used to help with the assessment of an adolescent struggling with psychiatric symptoms and migration issues, including various scales and questionnaires for the adolescent and his or her parents, an in-depth psychological evaluation, and collateral information from previous providers and others actively involved in the adolescent's life. An in-depth interview with the patient is critical and reveals details about the suicidal behavior from the perspective of the adolescent—the events, sensations, emotions, and interactions that surrounded the suicidal incident. It is also critical to obtain a detailed migration history that includes information pertaining to the

patient's premigration history, experience of migration, time spent in transit, degree of loss, traumatic experience(s), work and financial history, support systems, medical history, family's concept of illness, and level of acculturation, as well as the effect of migration on the patient's development. Similarly, an in-depth interview with parents of teenagers can help with understanding the patient, parental interactions, and parental perceptions surrounding the suicidal event (3, 7).

Remembering the structural barriers and cultural factors that affect Latino youths who are seeking help is also important. Some structural factors that affect youths include being new to the service-delivery systems, not understanding emergency room procedures, fears of being reported as being undocumented, and language barriers. Cultural factors that affect these youths include "familism, influences on a sense of individuality, and reverence to parents and family elders (8).

Culturally sensitive screening and assessment tools are critical to any services provided to a patient and his or her family to help reduce stigma, apprehensiveness, and engagement in treatment (2). Facilitating accurate assessment and improved utilization and effectiveness of services can be achieved by using approaches that address the need for validation, mutual support, and processing of common experiences (4).

Treatment and Prevention Strategies

Because of the strong cultural value of family, it is critical that treatment for immigrant adolescents focus on the family—parents and adolescents together and individually—to optimize the treatment. Some researchers have adapted therapies so that the intervention involves parents and their adolescent child. Family engagement is a recommended primary focus for treatment and prevention of suicidal behaviors in Hispanic females. The family in Hispanic culture holds a central position in individuals' lives, and thus it is important to treat the adolescent within the context of his or her family. However, individual therapy is also important (2).

Other treatment services can be considered, including medication management, group therapy, religious services, psychoeducation, and therapeutic mentorship. Preventive strategies that can be used include having primary care settings and school social workers and counselors to help identify adolescents at risk for suicidal behaviors, monitoring adolescents with immigrant parents, establishing support groups for adolescents and parents to discuss pressures, difficulties, and concerns, and establishing parent-oriented psychoeducational sessions about the developmental needs of adolescents and the natural tension that occurs during the acculturation process (2).

Conclusions

Migration involves changes and stresses that affect families collectively and individually. Acculturation and assimilation into mainstream U.S. culture is associated with health outcomes that are very complex and not well understood. Various groups are affected by the changes in different ways, and sometimes the changes can precipitate postmigration mental health issues. Culturally sensitive screening and assessment tools are critical to any services provided to a patient and his or her family to help reduce stigma, apprehensiveness, and engagement in treatment.

Dr. Zuber is a second-year child and adolescent psychiatry fellow at the Yale Child Study Center, New Haven, Conn.

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Acute Alcohol Withdrawal

Dylan P. Murray, M.D.

Case

“Mr. D” is a 37-year-old man brought to the emergency department by ambulance with his mother for mental status changes and agitation. According to his mother, the patient had a long-standing history of alcohol dependence and had experienced withdrawal seizures in the past. The mother believed that his last drink was 3 nights prior. Due to increasing agitation during transport, emergency medical services administered haloperidol (10 mg) and lorazepam (4 mg) intramuscularly but with little effect. In the hospital, the patient was tachycardic, tachypneic, and diaphoretic, and he shouted incoherently. His agitation continued to worsen despite an increase in the doses of lorazepam and haloperidol. Physical restraints were applied, and he was intubated and transferred to the medical intensive care unit.

Prevalence and Pathophysiology

Rates of hospital admissions and emergency department visits associated with alcohol use are high, and some studies have estimated that up to 20% of hospital inpatients and 40% of emergency department patients meet requirements for alcohol abuse or dependence. However, the prevalence of alcohol withdrawal in the U.S. population is relatively low (1).

Ethanol is active at two main receptor sites in the brain: GABA_A and *N*-methyl-D-aspartic acid (NMDA). By interacting with the GABA receptor throughout chronic exposure, ethanol enhances GABA's inhibitory effect without increasing the amount of neurotransmitter present. During ethanol withdrawal, the down-regulated inhibitory effect leads to increased neural activity. Ethanol has the opposite effect on NMDA receptors, causing up-regulation and increased

neural activity during withdrawal. The disruption in the balance of these neurotransmitters is likely responsible for the signs and symptoms of alcohol withdrawal syndrome, including seizures and delirium tremens (2).

Clinical Findings

The symptoms of alcohol withdrawal syndrome are best thought of as a spectrum that relates to a time course. Six to 12 hours after an individual's last alcohol consumption, minor symptoms, such as tremulousness, mild anxiety, gastrointestinal upset, headache, and palpitations, can be present. At about 1 day since the last drink, hallucinosis may set in, and after 24–48 hours without consumption, withdrawal seizures may occur. Withdrawal delirium (hallucinations, disorientation, agitation, tachycardia, fever, and diaphoresis) usually occurs 48–72 hours after the last consumption (3). Other clinical findings of alcohol withdrawal include hyperreflexia and tachypnea.

Pharmacologic Strategies

A Cochrane review showed that benzodiazepines protect against alcohol withdrawal symptoms, especially seizures (4). Benzodiazepines act as a substitute for ethanol activity at GABA receptors and decrease symptoms by increasing their affinity for GABA and augmenting inhibitory effects. In the Cochrane review, results comparing individual benzodiazepines did not show any statistically significant differences, although chlorthalidone tended to offer better control of symptoms. In emergency settings, lorazepam is commonly chosen because it is readily available in an intramuscular formulation.

There is little evidence base to guide dosing strategies, although symptom-triggered therapy (as opposed to a fixed dosing schedule) has been shown to be

more efficacious and to require less medication and cost (5). In symptom-triggered therapy, a standardized rating scale is used to assess the severity of symptoms, such as nausea, tremor, anxiety, and agitation, and benzodiazepine doses are determined by the results. This approach to dosing requires increased nursing staff but results in equally safe outcomes and decreased medication use and treatment duration (6).

Antipsychotics, such as haloperidol, are commonly used to treat the psychiatric symptoms that accompany alcohol withdrawal, such as hallucinations and agitation. Haloperidol is active at different neurotransmitter sites than benzodiazepines and thus should be used as adjunctive therapy. If haloperidol administration is required, evidence suggests that it is best to implement a fixed dosing regimen that continues after symptom resolution, since fluctuations in mental status are common (5). Cardiac monitoring is strongly recommended during haloperidol administration (especially if it is administered intravenously) due to its propensity to prolong the QTc interval.

While benzodiazepines (and haloperidol if necessary) are the mainstay of treatment, other pharmacologic agents are able to target withdrawal symptoms by acting at different receptor sites. Research in animal models and in humans has shown that brain levels of epinephrine drop during alcohol consumption but then rebound to above prealcohol exposure levels during withdrawal (7). Thus, the noradrenergic system is another important neurotransmitter system that is a possible therapeutic target. Studies have shown that clonidine, an alpha₂-agonist, provides benefit in that it reduces symptoms of sympathetic hyperactivity, but it does not offer protection against withdrawal seizures or delirium (7).

In addition to managing the overt signs and symptoms of alcohol withdrawal, it is

important to consider correcting concomitant dehydration, electrolyte imbalances, and vitamin deficiencies. If symptoms are so severe that extended periods of immobility are likely, prophylaxis for deep venous thrombosis should be considered.

Summary

Alcohol withdrawal syndrome is largely the result of the chronic dysregulation of GABA and NMDA receptor sites that is exposed by the abrupt cessation of alcohol intake. Signs and symptoms of alcohol withdrawal include tremor, mild anxiety, diaphoresis, disorientation, agitation, and seizure. Benzodiazepines are the mainstay of treatment for alcohol withdrawal due to their activity at GABA receptor sites, but pharmacologic agents active at

other neurotransmitter sites, such as anti-psychotics and noradrenergic agents, also play a role in reducing symptom burden. Other medical issues, including dehydration, electrolyte imbalances, and vitamin deficiencies, should also be considered.

Dr. Murray is a first-year resident in the Department of Psychiatry, Roy J. and Lucille A. Carver College of Medicine, University of Iowa Hospitals and Clinics, Iowa City, Iowa.

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Food-Triggered Panic Attacks

Chris A. Karampahtsis, M.D., M.P.H.

Panic attacks suggest a diagnosis of panic disorder, but alternate diagnoses must also be considered, especially when an unusual presentation confounds the clinical picture. By taking a careful history, it is possible to clarify the underlying problem and avoid diagnostic missteps. In this case report, a young woman presents with food refusal, dramatic weight loss, and panic attacks, creating a complex and potentially misleading presentation.

Case

“Miss B” is a 24-year-old single, Caucasian woman who was referred by the emergency department to the psychiatric inpatient unit for evaluation of depression and anxiety. She described a 3-month history of panic attacks and weight loss. The first attack occurred during a meal and included the abrupt onset of palpitations, shortness of breath, diaphoresis, tremors, paresthesias, lightheadedness, and a sense of losing control of her body. The attacks increased to as many as three daily and always occurred in situations involving food. The patient said that she avoided even the thought of food and hesitated to go out in public due to her fear of having another panic attack. She denied having any attacks in situations unrelated to food and also denied any past history of panic attacks. Although her food avoidance resulted in clinically significant weight loss, she denied having any intention of losing weight or fear of gaining weight. She showed no evidence of body image distortion and appeared appropriately concerned about the 20-lb weight loss. In addition to the panic attacks, she reported sleep onset delayed by 1 hour or more and admitted to anhedonia, fatigue, poor concentration, and poor appetite, all lasting at least 2 weeks. Recent psychosocial stressors included ongoing medical evaluation of abdominal pain, her parents’

divorce, a relationship breakup, and significant changes in her living situation.

The patient’s psychiatric history was consistent with dysthymia and generalized anxiety disorder, which were effectively treated by her family physician with escitalopram and alprazolam approximately 1.5 years prior to admission, but these medications were discontinued several months prior to admission. A trial of paroxetine was initiated a few weeks before admission but was not tolerated. She described social alcohol use and denied illicit drug use. Her family psychiatric history was remarkable for her mother having had postpartum depression and panic attacks and an uncle diagnosed with schizophrenia. Her medical history was notable for multiple recent emergency department visits related to abdominal pain and an extensive negative medical workup, which included laboratory studies, an ultrasound, and laparoscopy. Depot medroxyprogesterone therapy was initiated 2 weeks prior to her first panic attack in order to treat presumed endometriosis, and an oral contraceptive (levonorgestrel and ethinyl estradiol) was then discontinued. No other medications were reported.

On admission, Miss B presented as a frail, anxious young woman who described her mood as “low.” She was generally cooperative but easily fatigued. Mental status examination results were otherwise unremarkable, with no evidence of suicidality or psychosis. Her body mass index was 14.9 kg/m², and she was febrile, but her vital signs were otherwise stable. Physical and neurologic examinations were unremarkable except for suprapubic tenderness, muscle wasting, tremulousness, and weakness requiring assistance in sitting, standing, and walking.

Initial laboratory studies revealed elevated white blood cell count and urinalysis sug-

gestive of urinary tract infection. A urine drug screen was negative. Following internal medicine consult recommendations, blood and urine cultures were obtained, and the patient was started on intravenous fluids and antibiotics. Treatment with oral escitalopram and alprazolam was initiated. Miss B initially continued to refuse food and was fearful of having it brought to her room. She required a great deal of support and encouragement, initially verbalizing a preference for total parenteral nutrition, but was able to resume food consumption as the panic attacks began to subside. Two days after admission, she was transferred to a medical floor for treatment of pyelonephritis, iron deficiency anemia, dehydration, and malnutrition and was referred to an outpatient psychiatrist for further treatment following discharge. Unfortunately, she was lost to follow-up, and no further information is available about the course of her illness.

Discussion

On admission, Miss B’s presentation with acute weight loss and food avoidance suggested a diagnosis of anorexia nervosa, with which anxiety disorders, and thus panic attacks, frequently co-occur (1). Further evaluation revealed that she did not meet criteria for anorexia nervosa but was instead demonstrating a phobic avoidance of food due to recurring panic attacks triggered by food. A diagnosis of panic disorder under current guidelines requires the presence of at least some *unexpected* panic attacks. In this patient, no untriggered attacks were reported, and all attacks were described as having been brought on by a specific situation. DSM-IV-TR refers to this type of attack as a “situationally bound” or “cued” panic attack, since it “almost invariably occurs immediately after exposure to, or in

anticipation of, the situational cue or trigger” (2). This type of attack is commonly seen in anxiety disorders, especially phobias, which are characterized by bimodal onset, with the second peak occurring in the mid-20s, female predominance, and a familial pattern. On the other hand, both triggered and untriggered attacks are seen in panic disorder, and it is possible that some untriggered attacks had been misattributed or forgotten by the acutely ill patient. We found no reports of food-induced panic attacks in the literature. A specific phobia alone did not account for the rest of our patient’s clinical picture. In the context of chronic abdominal pain and unproven endometriosis, her symptoms suggested a possible somatoform disorder, but this was also ruled out. Malignancy was considered to be unlikely given the previous extensive negative medical workup, but evolving pyelonephritis undoubtedly contributed to the severity of her clinical presentation.

Miss B’s symptoms did meet criteria for major depressive disorder, with which panic attacks and panic disorder are frequently associated (3, 4). Onset of the patient’s panic symptoms 2 weeks after discontinuing an oral contraceptive and receiving an injection of medroxyprogesterone suggests a possible trigger for her presenting symptoms. The incidence of depression associated with depot medroxyprogesterone is reported to be 1.5% in standard prescribing information. Panic attacks per se have not been reported, and some evidence suggests that medroxyprogesterone may have a protective effect on panic and anxiety (5). Another long-term progestin-only form of contraception, levonorgestrel implants, has been reported to induce depression and panic (6). However, one study found that, relative to the use of levonorgestrel implants, lack of relationship satisfaction more accurately predicted depressive symptoms (7). An increase in panic also has been noted specifically with triphasic

oral contraceptive use (8). Interestingly, some case reports indicate that estrogen replacement therapy may contribute to the onset of panic disorder (9), while others indicate that estrogen replacement may dissipate panic (10). Although it is still unclear how progesterone or estrogen is related to the onset of depression and panic disorder, it appears possible that hormonal factors played a role in our patient’s presentation. It should also be noted that symptoms of depression and anxiety are commonly associated with diagnosed pelvic endometriosis (11).

Conclusions

The prominent physical complaints in the present case initially obscured the psychiatric diagnosis and delayed effective treatment. Consequently, the patient’s physical and emotional condition deteriorated, and her ability to function in daily life was impaired enough to require hospitalization. In cases of severe weight loss, in which insufficient evidence exists for a diagnosis of anorexia nervosa and a rigorous medical workup uncovers no evidence of physical pathology, we recommend that those who present with panic attacks be questioned carefully regarding food-related cue triggers.

Dr. Karampahtsis is a third-year resident in the Department of Psychiatry, Western Michigan University School of Medicine, Kalamazoo, Mich.

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Neuroleptic Malignant Syndrome in Patients With HIV in the Emergency Department

Danijela Ivelja-Hill, M.D.
Eileen Zhivago, M.D.
Maria del Pilar Trelles-Thorne, M.D.

Neuroleptic malignant syndrome is an uncommon and potentially fatal side effect of antipsychotic medications, characterized by severe rigidity, tremor, fever, altered mental status, autonomic dysfunction, and elevated serum creatinine phosphokinase and white blood cell count (1, 2). Patients with HIV are known to be more susceptible to developing neuroleptic malignant syndrome (2, 3).

We describe the cases of two female patients with known histories of HIV/AIDS who presented in the emergency department with acute psychosis. Both patients were treated with antipsychotic medications and subsequently developed neuroleptic malignant syndrome within 24–72 hours. Neuroleptic malignant syndrome was not anticipated by the emergency department physicians or by the psychiatrists in the emergency department setting. Increased awareness of this grave and potentially deadly complication of treatment with antipsychotic medications is needed.

Case 1

“Ms. A” is a 51-year-old single, African American woman with a history of schizophrenia, HIV, and opioid dependence as well as a history of known treatment noncompliance, except for treatment with highly active antiretroviral therapy and methadone. She presented to the emergency department with paranoid delusions. She was well known to our emergency department service and would usually present with paranoid persecutory delusions and agitation. She arrived acutely psychotic, with a delusion that a female neighbor was entering her apartment and looking at her, even through the walls. She was also agitated, verbally aggressive, and threatening.

In an attempt to control her symptoms, the patient was medicated three times over the course of 6 hours. The cumulative dose of medication was 3 mg of risperidone and 5 mg of haloperidol, along with 50 mg of diphenhydramine, 2 mg of benztropine, and 2 mg of lorazepam by mouth. Her psychotic symptoms improved, and she was discharged home only to return in 2 days with an acute change in her mental status, tremors of the upper extremities and face, and cogwheel rigidity. Her temperature was 100.5°F, and her heart rate ranged from 80 to 100 beats per minute. Neuroleptic malignant syndrome was strongly suspected. Her blood urea nitrogen level was 74 mg/dL (reference range: 7 mg/dL–21 mg/dL), and creatinine concentration was 2.4 mg/dL (baseline: 1.2 mg/dL; reference range: 0.6 mg/dL–1.1 mg/dL). The patient’s white blood cell count was 10,700 cells/mL (baseline: 2,300 cells/mL, reference range: 3,500 cells/mL–11,000 cells/mL), Her creatinine kinase level was 1,224 U/L (reference range: 30 U/L–200 U/L), and her CD4 count was 182 cells/mL (reference range: 400 cells/mL–1,500 cells/mL).

The patient was admitted to the medical unit, where a thorough medical workup excluded all infectious causes of fever and change in mental status. She was treated with supportive measures, and cessation of antipsychotic medication was implemented. Symptoms of neuroleptic malignant syndrome resolved within a week. While initially compliant with treatment, the patient started to become easily agitated and at times became violent toward the staff and had persecutory delusions. She was transferred to the inpatient psychiatric unit for further management.

After 2 months of a very complicated hospital course, she was eventually stabilized and discharged home.

Case 2

“Ms. B” is a 35-year-old single, African American woman with a history of bipolar disorder and HIV. She was brought to the emergency department from jail after 5 days of incarceration. No information was available as to whether she had been medicated while incarcerated. Her valproic acid level was 12 µg/L (reference range: 50 µg/L–100 µg/L) on arrival. However, she stated that she was not taking any medications prior to incarceration. She presented as disorganized with incoherent speech and had blunted and at times silly affect and agitation. She was medicated with 5 mg of haloperidol, 2 mg of lorazepam, and 50 mg of diphenhydramine intramuscularly once. Overnight, she became lethargic and rigid. Her maximum temperature was 103.6°F, and her heart rate ranged from 92 to 143 beats per minute. Her white blood cell count was 6,700 cells/mL, her creatinine kinase level was 2,588 U/L, and her CD4 count was 90 cells/mL.

The patient was admitted to the medical unit and underwent an extensive workup that excluded infectious causes for her condition. All medications were discontinued, and supportive treatment was administered with the addition of bromocriptine (2.5 mg p.o., b.i.d. for 5 days). Initially, she was very confused, disoriented, and irritable and had to be placed in soft restraints several times to prevent pulling her intravenous lines. However, she quickly improved, and within 5 days she was coherent, logical, and euthymic and did not have any psychotic symptoms. After 7 days, she was discharged home with outpatient follow-up.

Discussion

Ever since neuroleptic malignant syndrome was first described by Delay and colleagues in 1960, it has been a diagnostic and treatment challenge for clinicians (4). It is underdiagnosed outside of psychiatry because its manifestations may mirror symptoms caused by infections and neoplasms. A central dopaminergic hypoactivity has been suggested as a possible trait marker of the disorder (5). Sixty-six percent of cases will develop within the first week of antipsychotic treatment and 16% within the first 24 hours, with the remainder of cases developing sometime within the first month of treatment (2). Thus, neuroleptic malignant syndrome can develop in a very short period of time, even while a patient is still being managed in the emergency department.

Risk factors for neuroleptic malignant syndrome appear to include high potency antipsychotic medications, such as haloperidol and risperidone, rapid-dose increasing and/or intramuscular administration, loading rate of the neuroleptic, concomitant administration of anticholinergic medication, suboptimal electrolyte/hydration status, exhaustion, increased psychomotor activity, prior episodes of the disorder, and medical illness (2, 6). These risk factors pertain to the general population as well as to HIV-stricken patients.

Our patients had low CD4 counts, which correlate clinically to the severity of HIV/AIDS. History of affective disorder, alcoholism, and crack/cocaine use appear to increase the risk for neuroleptic malignant syndrome in the HIV population (6).

Patients receiving antipsychotics should therefore be medicated cautiously, with low doses and slow dosage increases, and closely monitored for development of any symptoms associated with neuroleptic malignant syndrome. Patients with HIV appear to be very susceptible to the disorder, even after small and/or single doses of antipsychotic medications are administered. The proposed mechanism of this

susceptibility is decreased dopamine levels throughout the brain, particularly in the basal ganglia, caused by HIV-induced neurodegeneration (5, 7). HIV affects the brain through neuroinflammation. The primary CNS cell types vulnerable to HIV infection are the parenchymal microglia and macrophages. Given the lack of evidence for significant neuronal infection, it is assumed that neuronal damage and death in HIV/AIDS is an indirect result of HIV infection of other cells in the brain. Changes may be present in any CNS area, particularly in the basal ganglia and central white matter, as well as in neocortical gray matter and the brainstem and cerebellum (8).

The first scenario described above also presents the daunting clinical problem of when it is safe to restart antipsychotics in a patient with a history of neuroleptic malignant syndrome. The currently accepted guidelines are to wait 2 weeks and restart the lowest possible dose of an atypical antipsychotic (2). Nevertheless, in the HIV population, a particularly cautious approach should be considered.

Prior to the neuroleptic malignant syndrome episode, one of our patients was taking highly active antiretroviral therapy medications, and the other was not. Some investigators have proposed that treatment with highly active antiretroviral therapy is not neuroprotective; it only shifts the primary site of neuroinflammation from the basal ganglia to the hippocampus and surrounding entorhinal and temporal cortex (8). Therefore, even patients receiving highly active antiretroviral therapy could be more sensitive to neuroleptic medications, especially because many protease inhibitors can increase antipsychotic levels by blocking cytochrome P450 enzymes, particularly risperidone (9).

Early recognition of neuroleptic malignant syndrome is very important because early treatment improves the outcome. HIV patients who arrive in the emergency department with acute changes in their mental status receive a full battery of extensive medical tests, but neuroleptic

malignant syndrome is rarely considered, even after all other causes have been excluded. The role of psychiatrists is instrumental in educating colleagues about the possibility that this syndrome can develop rapidly in the HIV/AIDS population and in placing the syndrome higher on the differential diagnosis list for this vulnerable population.

Drs. Ivelja-Hill and Zhivago are fourth-year residents, and Dr. del Pilar Trelles-Thorne is a third-year resident in the Department of Psychiatry, University of Medicine and Dentistry of New Jersey, Newark, New Jersey.

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Hypersexual Disorder: A Clinical Review for Psychiatry Residents

Iman Parhami, M.D., M.P.H.

The Sexual and Gender Identity Workgroup is considering including behaviors such as sexual affairs, sexual promiscuity, and online sexual indiscretions as part of a psychiatric condition in DSM-5, labeled hypersexual disorder (1). Recognizing the signs and symptoms of this disorder can be confusing at times because the line between normative and pathological behaviors is not well defined (2). For that reason, the reliable classification is important in facilitating communication among clinicians and patients, as well as in providing a framework for delivering effective therapeutic interventions. To increase familiarity of hypersexual disorder among psychiatry residents, this article will review diagnostic criteria, clinical presentation, and treatment.

Diagnostic Criteria

It is difficult to diagnose hypersexual disorder (also referred to as sexual addiction or sexual compulsivity) solely based on sexual behavior. It is necessary to gather as much information about the presenting problem from as many reliable sources as possible and then differentiate those symptoms from more parsimonious explanations of the chief complaint. Clients that potentially have hypersexual disorder should be evaluated in the context of several other factors, including the extent to which sexual activity (including preoccupation) 1) disrupts personal, family, social, or vocational pursuits; 2) causes significant personal distress to self or others; 3) poses the risk or potential for significant physical or emotional harm to self or others; 4) is uncontrollable or resistant to change (e.g., the patient feels out of control or unable to reduce or change the behavior); and 5) is not better accounted for by an alternate psychiatric diagnosis and is not substance induced (2).

According to the proposed criteria for DSM-5, individuals may be determined

to have hypersexual disorder diagnosis when they have recurrent and intense sexual fantasies, sexual urges, and sexual behavior for at least 6 months and satisfy at least four of the following symptoms: 1) excessive time is consumed by sexual fantasies and urges and by planning for and engaging in sexual behavior; 2) repetitively engaging in these sexual fantasies, urges, and behaviors in response to dysphoric mood states (e.g., anxiety, depression, boredom, or irritability); 3) repetitively engaging in sexual fantasies, urges, and behaviors in response to stressful life events; 4) repetitive but unsuccessful efforts to control or significantly reduce these sexual fantasies, urges, and behaviors; and 5) repetitively engaging in sexual behavior while disregarding the risk for physical or emotional harm to self or others (3).

After hypersexual disorder is suspected, clinicians should inquire about relevant domains related to high-risk sexual behavior, such as the number of recent sexual partners and whether the individual used protection during sex. Other risks for physical or emotional harm should also be considered, for example, locations where the individual masturbates (i.e., in a public setting, workplace, etc.) and whether the individual enters high-crime neighborhoods to engage with a commercial sex worker. Finally, clinicians should also be aware of the social consequences related to hypersexual disorder, such as jeopardized romantic relationships and problems at work caused by sexual behavior (1, 4–6).

A number of self-report measures capture the associated characteristics of hypersexual behavior (7, 8), including the Hypersexual Behavior Inventory (9). This scale captures aspects of hypersexual disorder such as engaging in sex in response to stress (e.g., “Doing something sexual helps me cope with stress”) or to dysphoric mood (e.g., “I turn to sexual activities

when I experience unpleasant feelings”) or multiple unsuccessful attempts to diminish or control sexual thoughts, urges, and behaviors (e.g., “Even though I promised myself I would not repeat a sexual behavior, I find myself returning to it over and over again”) (9).

In the scientific literature, symptoms of hypersexual disorder have been conceptualized to be part of obsessive-compulsive disorder (OCD), as well as impulse control and addictive disorders (10). However, there are inconsistencies when categorizing hypersexual disorder solely based on one of these groups of disorders. For example, unlike hypersexual disorder, OCD does not involve reward, and impulse control disorders (e.g., trichotillomania) do not involve planned activities to achieve goals. In addition, the standard treatments for OCD (exposure and response prevention) may differ from those thought to be effective for hypersexual disorder. Until more research explores the pathophysiological correlates of hypersexual disorder, using a theory-neutral term that can cut across these categories is more advantageous (10).

Clinical Presentation

Usually, hypersexual disorder is encountered in the clinic when individuals seek help as a result of a loved one’s ultimatum after an exposed affair or when the disorder is diagnosed inadvertently after presentation of another comorbid condition. Common comorbid psychopathologies include mood, anxiety, attention-deficit, and substance-related disorders (11, 12). Personality characteristics, such as boredom proneness (13), impulsivity and shame (14), interpersonal sensitivity, alexithymia, loneliness, and low self-esteem, have also been observed in association with hypersexual behavior (15, 16). Two potential cases of hypersexual disorder are presented below.

Potential Cases

“John” is a 65-year-old traveling salesman, referred by his new primary care physician for treatment of possible hypersexual behavior following his desire to refill his Viagra prescription. He takes Viagra daily and reports having sex with his wife at least once per day. He has been happily married for 20 years and has sustained a salesperson job for 10 years. He reports 10 “one nighters” on business trips in the past year. His wife is aware of these “one nighters” and accepts this type of arrangement. His sexual behavior has never interfered with his health, work, or family life. He is in optimal physical health and does not show signs of any other psychiatric condition.

“Max” is a 21-year-old single, male college student, referred by school police for masturbating in the library. Although this is his first citation, he reports masturbating more than 10 times in the school library in the past month. He also reports engaging in unprotected sexual activity with random young women at fraternity parties and with prostitutes a few times per month. He has had 30 sexual partners in the past year (and never uses condoms). His schoolwork is suffering because he says he cannot concentrate without thinking about sexual fantasies. He is currently HIV-positive due to sexual transmission. He reports using sex (with a partner or individually) to deal with stressors and depression. He admits that once he has the urge to have sex, he does not have control over his actions and needs to either masturbate or have sex wherever he is (including once when he masturbated while driving a car).

Discussion of Potential Cases

Solely based on the amount of sexual activity, John may be presumed to have hypersexual disorder on first impression. However, after exploring a more elaborate history of both patients, it is evident that John has not suffered any consequences or repercussions from his sexual behavior, while Max has. Compared with John, Max engages in risky sexual behavior (unprotected sex, including sex with prostitutes) and has experienced harm from his sexual behavior (i.e., HIV-positive status, poor schoolwork, cited for masturbation in the school library, etc.). In addition, Max reports a loss of control over his sexual fantasies, urges, and behaviors. Given this information, Max meets the proposed criteria for hypersexual disorder, and John does not.

Treatment Approaches

The goal of treatment should be to arrest hypersexual behavior, address underlying issues that precipitate or perpetuate problematic sexual behaviors, and help enhance emotional regulation and stress coping strategies. Although empirical studies of

hypersexual disorder are scant, a few case studies and nonrandomized reports have documented successful treatment for patients with the disorder, including those that involve cognitive-behavioral therapy, psychodynamic psychotherapy, 12-step programs, couples therapy, and psychopharmacological treatments (7, 17). For instance, relapse prevention techniques can assist with anticipating and coping with relapse, and imaginal desensitization can be effective in reducing compulsive sexual behaviors (17). Self-help groups (i.e., Sex and Love Addicts Anonymous, Sex Addicts Anonymous, Sexaholics Anonymous, and Sexual Compulsives Anonymous) are intended to provide a supportive atmosphere, in which individuals with hypersexual disorder can focus on reducing problematic behavior and help each other during the recovery process (17). Pharmacological treatments include selective serotonin reuptake inhibitors, which may reduce comorbid anxiety and depressive symptoms, and opioid antagonists (e.g., naltrexone), which can reduce unwanted behaviors in urge-driven disorders (18, 19).

Conclusions

Research on hypersexual disorder is rapidly emerging because the disorder is being considered for inclusion in DSM-5. Given the significant harm to individuals, families, and society that is correlated with hypersexual disorder, psychiatry residents should be aware of the proposed diagnostic criteria for this disorder. When assessing a potential case of hypersexual disorder, the amount of sexual behavior should not be the only detriment explored for diagnosis. Other components include whether the behavior causes distress and disrupts personal, family, social, or vocational pursuits. Individuals with hypersexual disorder should also be screened for comorbid anxiety, mood, and substance-related disorders, as well as sexually transmitted diseases.

At the time this article was accepted for publication, Dr. Parhami was a postdoctoral research fellow in the Department of Psychiatry and Biobehavioral Sciences, University of California, Los Angeles, studying pathological gambling and other behavioral addictions.

The author thanks Drs. Rory Reid and Timothy W. Fong for their guidance and assistance with this article.

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The Editors of *The American Journal of Psychiatry* have developed a special mobile-optimized website that displays a single bit of *Clinical Guidance* every day gleaned from research published on the pages of the *Journal*. Users can click through to the main article or explore an archive of all previously prepared *Clinical Guidance* pieces arranged by topic.

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Bethanechol for Antidepressant-Associated Sexual Dysfunction

Jonathan R. Scarff, M.D.

Drs. Shapov and Forcen recently reviewed the management and treatment of selective serotonin-reuptake inhibitor (SSRI)- and selective norepinephrine reuptake inhibitor (SNRI)-induced sexual side effects in patients with depression (1). Monoamine oxidase inhibitors and tricyclic antidepressants may cause similar dysfunction. However, there is evidence for the effectiveness of bethanechol in alleviating this problem.

In daily doses ranging from 12.5 mg to 50 mg, bethanechol eliminated anorgasmia in a woman receiving amoxapine and reversed erectile dysfunction in two men, one receiving isocarboxazid and the other receiving tranlycypromine (2). When taken 30 minutes to 2 hours prior to intercourse, 10 mg–20 mg of bethanechol enabled patients receiving either imipramine or protriptyline to obtain erections (3, 4). In a double-blind placebo-controlled crossover study, 10 male patients receiving clomipramine regained capacity for orgasm after taking 40 mg of bethanechol 45 minutes prior to intercourse (5).

Bethanechol may alleviate sexual dysfunction by reversing anticholinergic activity. More likely, its selective muscarinic agonism potentiates the adrenergic system in which the receptors have been blocked by an antidepressant (6). Because of its cholinergic activity and adrenergic potentiation, bethanechol may induce flushing, intestinal cramping, gastric secretion, bronchoconstriction, arrhythmia, headache, urinary urgency, and sympathomimetic effects. Contraindications include asthma, uncontrolled hypertension or hyperthyroidism, peptic ulcer disease, intestinal obstruction, and coronary artery disease (2). Evidence for efficacy is largely limited to case reports. There is only one report of efficacy in women, and the drug is unexplored in patients receiving newer antidepressants. However, bethanechol may be worth considering as another treatment option for reversing sexual adverse effects.

Dr. Scarff is a fourth-year resident in the Department of Psychiatry and Behavioral Sciences, University of Louisville, Louisville K.Y.

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TEST YOUR KNOWLEDGE

In preparation for the PRITE and ABPN Board examinations, test your knowledge with the following questions.
(answers will appear in the next issue)

In preparation for the PRITE and ABPN Board examinations, test your knowledge with the following questions (answers will appear in the next issue).

This month's questions are courtesy of David Goldsmith, M.D., a first-year resident at Emory University School of Medicine, Atlanta.

Question #1

Glucocorticoids are prescribed for a variety of diseases and are known to cause a number of somatic and neuropsychiatric side effects. Which of the following is a true statement regarding the neuropsychiatric complications of glucocorticoid use?

- A. Psychiatric side effects of glucocorticoids are only seen after administration of increasingly large doses of steroids.
- B. Neuropsychiatric side effects are typically seen after months of corticosteroid use and not before.
- C. Neuropsychiatric side effects of glucocorticoids are only seen with systemic administration of the medication.
- D. Dosage is the most significant risk factor for neuropsychiatric side effects of glucocorticoid therapy.

Question #2

Which of the following has been shown to be effective in augmenting and/or accelerating treatment in major depressive disorder?

- A. Triiodothyronine (T₃)
- B. Thyroxine (T₄)
- C. Thyroid-stimulating hormone
- D. Iodine supplementation

ANSWERS TO NOVEMBER QUESTIONS

Question #1.

Answer: C Intense feelings of worthlessness

Feelings of worthlessness are not explicitly part of the criteria for post-traumatic stress disorder (PTSD) as outlined in DSM-IV-TR (1).

Reference

1. American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders, 4th ed (Revised). Washington, DC, 2000

Question #2

Answer: B Prazosin

A majority of people with PTSD have substantial problems with sleep disturbances (1). There is increasing evidence that PTSD-associated nightmares may be successfully treated with prazosin (2). Prazosin has also demonstrated better long-term effectiveness, compared with quetiapine, in reducing PTSD sleep disturbances (3). There is limited evidence for the use of bupropion or melatonin in treating PTSD-associated nightmares.

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- *Please direct all inquiries and submissions to Dr. Vahabzadeh: arshya.vahabzadeh@emory.edu.

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- 6. Letters to the Editor:** Limited to 250 words (including 3 references) and three authors. Comments on articles published in *The Residents' Journal* will be considered for publication if received within 1 month of publication of the original article.
- 7. Book Review:** Limited to 500 words and 3 references.

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March 2013

Section Theme: Women's Mental Health
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April 2013

Section Theme: Military Psychiatry
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May 2013

Section Theme: DSM-5
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June 2013

Section Theme: Psychiatry and Social Justice
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