Letters to the Editor

Re: From Chlorpromazine to Clozapine—Antipsychotic Adverse Effects and the Clinician’s Dilemma

Dear Editor: Dr Abidi and Dr Bhaskara have accurately described new concerns arising from the use of atypical antipsychotics (1). The pharmacotherapy of schizophrenia remains an ongoing challenge for researchers and clinicians. The atypicals cause fewer extrapyramidal side effects (EPSEs) and tardive dyskinesia (TD), but there is growing concern regarding their significant long-term adverse metabolic and cardiac effects. These risks may be comparable to the EPSEs and TD associated with typical antypsychotics. Since atypical antipsychotics are all equally effective, the choice of drug for any given patient is still determined by their relative adverse-effect profile. Thus early identifica
tion of patients at high risk for obesity, diabetes, and cardiac disease is essential and requires increased monitoring and management (1).

Freedman (2) has recently described the current knowledge about the psychopharmacologic treatment of schizophrenia, a chronic and debilitating psychotic mental disorder wherein the similar therapeutic response among the new drugs emphasizes that choice be determined by the side effects profile.

The suffering of schizophrenia patients, as well as the disorder’s economic and social costs, is always underestimated. Schizophrenia has a considerable impact on patients, their families, and the health care system (3). Most patients experience repeated episodes with worsening outcomes: 9% suffer lasting impairment, and 43% endure increasingly severe symptoms with no periods of complete remission (4). Schizophrenia patients face impoverished lifestyles, finding it difficult to secure paid employment and to hold on to their jobs when they get them. The development and more widespread use of atypical antipsychotics may lead to a short-term increase in the proportion of the total cost of schizophrenia attributable to medications. Conversely, their use may lead to reduced hospitalization and allow patients to work and lead a normal social life, apart from the still-unknown cost of dealing with metabolic and cardiac side effects. Early diagnosis and precise treatment with the safest possible antipsychotics are key elements for lowering the social and economic burden of schizophrenia, but clinicians should be aware of the new side effects associated with long-term use of atypical antipsychotics.

References

Antonio E Nardi, MD
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Reply: From Chlorpromazine to Clozapine—Antipsychotic Adverse Effects and the Clinician’s Dilemma

Dear Editor: We thank Dr Nardi for his comments, which underscore the points we made and the challenges we face in treating schizophrenia.

In evaluating the personal, social, and economic burden of schizophrenia, we need to consider not only the immediate gains but also the long-term sequelae of pharmacotherapy.

Regulatory bodies such as Canada’s Therapeutic Products Directorate, the US Food and Drug Administration, and other similar agencies usually base their approval for antipsychotic drugs on a few Phase III clinical trials, where the study drug or treatment is given to large groups of people (approximately 1000 to 3000) to confirm its effectiveness, monitor its side effects, compare it with commonly used treatments, and collect information that will allow it to be used safely. In most cases, these are short-term, double-blind, placebo-controlled trials. Further, in seeking approval, pharmaceutical firms are not obligated to submit all data (both positive and negative) from Phase III clinical trials. Therefore, continued post-marketing vigilance and surveillance play a crucial role in evaluating long-term risks and benefits.

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Autism: Multiple Genes Acting on a Distributed Neural Target?

Dear Editor: Genetic research on the autistic spectrum disorders (ASDs) needs an informative endophenotype (1), that is, a common denominator that would distinguish brain functioning in the ASDs from normal functioning. In physiological functioning, simple reproduction of inputs takes place in primary sensorial areas, and integration of congruent unimodal data into more significative multimodal ones is enacted in associative areas (2). Higher levels of categorization and understanding are progressively reached, and a final choice among many (often emotionally charged) options guides voluntary and adaptive actions. The amount of data to be computed increases exponentially through all those stages. Enlarging webs of neural organizations cooperate for the purpose, continuously and smoothly changing their configuration and distribution of activation. It is difficult to conceive this kind of functioning without the intervention of a modulatory system (3) to integrate and set priorities among incoming and outgoing data. In a recent Positron Emission Tomography study, Hall and others found that individuals with autism are less prone to associate inputs from different sensorial modalities and that they preferentially allocate attentional resources to partial unimodal (both positive and negative) from Phase III clinical trials. Therefore, continued post-marketing vigilance and surveillance play a crucial role in evaluating long-term risks and benefits.

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References
comprehension tend to concentrate on stereotyped play—probably as a result of the unintegrated hyperfunctioning of discrete brain areas—to the exclusion of parallel and contextually appropriate perceptions and behaviours, such as turning their heads toward a person who is calling them by name. Analogously, a high-functioning and verbal 6-year-old girl with autism avoids scanning the eyes–nose–mouth zone of an interlocutor while trying to express herself verbally, seemingly in the attempt to make mental space available for language processes. In her late teens, the same girl will not be able to maintain multiple mental activities in an activated and easily accessible state—activities such as holding in mind all the meanings of a homograph while reading a phrase in which that homograph is embedded and which would restrict lexical choice to only a single meaning. Theory of mind tasks in a social environment probably exceed the maximum synchronous mental load tolerable by most persons with autism; only a more parsimonious and less efficient piecemeal computing of social inputs in a structured environment is possible. I suggest that reducing the amplitude of synchronously elicitable mental functions with relative preservation of discrete processing is a basic endophenotype of the ASDs, although admittedly a difficult one to use for diagnosis and genetic studies. In ASDs, multiple mutating genes may have cumulative damaging effects on a modulatory circuit similar to that which Andreasen postulated to be altered in schizophrenia (5). The clinical variability of ASDs may derive from the number of altered genes, the distribution of the damage to the integrative network, the degree and prevalence of the consequent hemispheric dysfunction, the presence of comorbidities (the most common being mental retardation), the temperament of the affected individual, and the compensatory mechanisms at work. Neuroplasticity would account for partial reversibility of the above-described dysfunc-

tion and explain the improvements that we detect in most individuals with autism as they become older, both spontaneously and under the influence of educational treatments on brain chemistry and structure.

References


Silvio Loddo, MD
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Recurrent Paroxetine-Induced Hyponatremia

Dear Editor: Selective serotonin reuptake inhibitors (SSRIs) have been implicated in the etiology of hyponatremia. Early- and delayed-onset hyponatremia have both been reported (1). We describe a case of early-onset, recurrent hyponatremia initially arising from paroxetine treatment and later, from paroxetine extended release.

Case Report

Mrs R, aged 71 years and married, had a long history of mood and anxiety symptoms. In 2000, her internist started her on paroxetine at a dosage of 20 mg daily. A week later, she was seen in the clinic for increasing confusion, malaise, and “not feeling good.” Her sodium level was found to be 120 mmol/dL, and she was admitted to the hospital. Paroxetine was thought to be the offending agent and was stopped. Her condition improved, and she was discharged from the hospital 2 days later, with serum sodium of 129 mmol/dL.

About 3 years later, her internist rechallenged her with paroxetine, this time prescribing the extended release preparation at 12.5 mg daily, titrated later to 25 mg daily. Approximately 10 days after the titration, she was seen in the clinic with symptoms of “not feeling well” and “depression” marked by fatigue and anxiety. Her sodium level was found to be 123 mmol/dL. She was rehospitalized, and upon review, it was found that her sodium levels had been fairly stable after she stopped taking paroxetine in 2000. A workup showed increased sodium excretion and reduced serum osmolality consistent with the syndrome of inappropriate antidiuretic hormone secretion (SIADH). She was cross-titrated to bupropion, and her sodium level at the time of discharge was 126 mmol/dL, which later improved to 133 mmol/dL on day 4 after discharge.

Discussion

Hyponatremia from SSRIs has been recognized to be more common in older patients. Stedman and others believe that there is no genetic propensity and that this effect is not dosage dependent (2). Regardless of the etiology, the effects can range from anhedonia, fatigue, and tiredness to confusion, coma, and permanent neurological injury. The literature indicates that SIADH can either manifest itself in days or take months to surface (1).

In our patient, the timeline links paroxetine to SIADH. The onset within a week of the first trial and within 2 weeks of the second trial, together with a sudden resolution only a day or 2 after discontinuation or dosage reduction leaves little else to suspect. Our case shows that a different formulation of the same drug can result in a different presentation of SIADH in the same patient.

Although hyponatremia is not common, it appears that its presentation can vary widely in different patients and even in the same patient at different times. Given its serious consequences, clinicians need to be vigilant to diagnose the condition in time and to reverse it promptly, once diagnosed.

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References


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Spontaneous Orgasm Started With Venlafaxine and Continued With Citalopram

Dear Editor: Although antidepressants have been commonly associated with anorgasm (1), few case reports highlight unusual sexual responses, including spontaneous orgasm with antidepressants (2). I report the case of a patient who experienced spontaneous orgasm starting with venlafaxine treatment and continuing with citalopram.

Case Report
Mrs A, aged 48 years and married, was admitted as a psychiatric outpatient with depressive symptoms. She had been diagnosed with recurrent severe major depression with melancholic features. She had been unsuccessfully treated for over 2 years with combined moclobemide 900 mg daily and olanzapine 10 mg daily. When she applied to our clinic, her Hamilton Depression Rating Scale (HDRS) score was 34, despite this treatment protocol. She began to take venlafaxine extended release 75 mg daily. At the end of week 1, the dosage was increased to 150 mg daily. At the end of 3 weeks, she recognized spontaneous orgasms occurring 4 to 5 times daily. She stated that she felt strain because of them. Her complaints continued in the fifth week, and venlafaxine was ceased. Her spontaneous orgasms decreased 2 weeks after she stopped taking venlafaxine. Then, she started taking citalopram 10 mg daily, increased to 20 mg daily after 1 week. The side effects returned to their past levels. Because she had either continued depressive symptoms or similar side effects from 2 different groups of antidepressants, drug treatment was given up, and electroconvulsive therapy (ECT) was started. After ECT, she recovered from both spontaneous orgasms and depression.

This is the first report of venlafaxine-induced spontaneous orgasm in women, and this is also the first report of spontaneous orgasm related to citalopram. Because this patient’s HDRS score was 25 and there were no symptoms of mania, her sexual experience did not seem to be owing to drug-induced hypomania. Further, this side effect occurred simultaneously with venlafaxine use and decreased after this drug was given up. Although the mechanism of this adverse effect is not known exactly, venlafaxine’s central effect on serotonin levels and peptides such as endorphine are thought to be related to it (3). It is not clear how this side effect disappeared. Improvement may have been owing solely to stopping citalopram or to ECT treatment. To my knowledge, nothing in the literature discusses how ECT affects sexual function. Therefore, this issue is worthy of research.

References

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Venlafaxine-Induced Mania

Dear Editor: In bipolar disorders, the shift to mania as a result of using antidepressants constitutes a great trouble for clinicians. It has been known for a long time that tricyclic antidepressants, monoamine oxidase inhibitors, selective serotonin reuptake inhibitors, and even bupropion can cause mania or hypomania (1,2). In clinical studies, venlafaxine, which acts through the blockage of serotonergic or noradrenergic receptors, proved to be an efficient, reliable, and rapidly effective drug that could be used without any problem to treat bipolar depression (3). The literature reveals only a few cases of venlafaxine-induced mania and hypomania (5,7). However, in the case we report, the treatment dosage of venlafaxine resulted in mania.

Mr F, aged 38 years, had a diagnosis of bipolar disorder followed in different centres for 16 years. Within this period, he experienced 2 manic and 2 hypomanic attacks. Two years prior to this report, he was taking lithium 1200 mg daily for prophylaxis but gave up taking the drug because he felt healthy. Approximately 1 month before presenting, he began to feel valueless and bad, without any reason. He did not want to do anything, slept all day, and never spoke. He had frequent thoughts that suicide would solve all his troubles but felt it was something he could not do. His appetite decreased, and he lost 4 to 5 kg within this period. Owing to these problems, he was admitted to an outpatient clinic. A general practitioner prescribed venlafaxine 75 mg daily. After 10 days, his sleep gradually began to decrease, and he started to speak excessively. He was feeling euphoric. He asserted that he was very handsome and enjoyed aggressive and angry behaviours. He was immediately taken to our emergency service and hospitalized with the diagnosis of bipolar disorder, manic episode. After his relatives provided a history, venlafaxine was stopped, and haloperidol and carbamazepine as a mood stabilizer were prescribed. His clinical picture improved rapidly within the first few days. He stayed in our clinic for a week and was then discharged from the hospital. Thereafter, he and his relatives were informed about the clinical presentation and prodromal symptoms of mania and depression.

The elimination of mania symptoms shortly after stopping antidepressant treatment suggests that venlafaxine could have caused the development of mania. Of course, it is also possible that the patient’s nonuse of a mood stabilizer while using an antidepressant may have led to the development of mania. It is not clear whether these shifts depend on the antidepressant dosage and on the length of use. The literature contains contradictory reports regarding this issue (5–7). In our case, the manic shift was experienced as a result of venlafaxine 75 mg daily taken for only 10 days.

It should be remembered that it is necessary to get detailed information about every patient applying for depression treatment. The risk of bipolar disorder is especially high in early-onset acute depression, chronic depression, and seasonal pattern depression, as well as in patients suffering from hyperthymic–cyclothymic temperament and in those having a family history of bipolar disorder. These patients should be followed closely during the first stage of treatment (8). What really constitutes a great trouble for us is the treatment of a bipolar depressive episode, as in our case. For these patients, mood stabilizers should be given; if the patient already uses mood stabilizers, their dosage should be increased. If the treatment fails, antidepressant treatment may be applied, but the patient should be followed regularly at short time intervals (9).
Episodic Ataxia vs Somatization Disorder

Dear Editor: The presentation of unexplained ataxia requires a broad differential including somatoform disorders. We report a case of episodic ataxia type 2 (EA-2) that was misdiagnosed as a possible somatization disorder.

EA-2 is a rare disorder that results from a multitude of mutations in the CACNA1A gene. CACNA1A codes for calcium channel proteins and is heavily expressed in the cerebellum. Patients with EA-2 generally present before age 20 years with ataxic episodes associated with vertigo, weakness, and interictal nystagmus. Such episodes may last hours to days (1). A positive family history may aid in the diagnosis; however, de novo mutations are also known to occur (2), making genetic testing the only definitive diagnosis. Acetazolamide is considered the treatment of choice for EA-2, but not all patients respond to the drug (3).

Case Report

Ms C is a single Ghanaian woman, aged 24 years, currently attending fashion school. She was admitted to hospital after an abrupt onset of dizziness, limb ataxia, and disequilibrium that resulted in a fall. Ms C experienced these symptoms every 2 to 3 months. They persisted for 1 to 7 days before resolving spontaneously. She recalled suffering from these disabling episodes since age 11 years and attributed them to her sickle cell trait.

The psychiatry service was consulted to assess whether a somatiform disorder might be the cause of her ataxia. Although Ms C had several neurological complaints, she did not endorse any gastrointestinal, pain, or sexual symptoms necessary for the diagnosis of somatization disorder. While she had recently moved out of her family’s home following a deterioration of the relationship with her father and stepmother, there was no clear association between stressors in her life and the onset or exacerbation of her symptoms—a criterion required to diagnose conversion disorder. Her family did not identify any history of body dysmorphic disorder, hypochondriasis, or pain disorder. Ms C expressed concern about missing several classes in fashion school and was worried about falling behind. There was no indication of personality disorder or obvious secondary gain that would suggest a factitious disorder of malingering.

Ms C reported being raped twice in the past 2 years, but she did not meet the criteria for posttraumatic stress disorder or another anxiety disorder. She denied any depressive and psychotic symptoms or drug and alcohol use and had no other psychiatric history. At this point, we concluded that an Axis I disorder could not explain her symptoms, and the medical team continued to seek a medical cause to explain her presentation.

On physical examination, Ms C had limb ataxia, dizziness, gait unsteadiness, and diplopia on upward gaze. A noncontrast CT, EEG, electromyography, and ECG were all normal. Relevant laboratory tests were all within normal limits. Subsequently, a diagnosis of EA-2 was entertained, and Ms C failed a trial of acetazolamide. She was discharged from the medical ward without a definitive cause for her symptoms and no psychiatric follow-up.

Discussion

The above case helps emphasize the need to rule out somatoform disorders in patients with neurologic complaints. Despite nonconfirmatory medical investigations, both the medical and psychiatric teams should continue to search for genetic causes of her ataxia, such as EA-2.

References


Mirtazapine for Charles Bonnet Syndrome

Dear Editor: Charles Bonnet syndrome (CBS) is characterized by complex visual hallucinations in psychologically normal people; it is usually seen in elderly people in the context of ocular pathology causing visual deterioration (1). The main hypothesis is that these hallucinations represent release phenomena attributable to deafferentation of the visual association areas of the cerebral cortex that leads to a form of phantom vision. Most intriguingly, unlike visual hallucinations associated with psychiatric disorders, patients with CBS have insight and report the hallucinations as nonthreatening. Several modalities have been tried to treat this condition, including valpromide (2), reperidone (3), carbamazepine (4), melperone (5), valproate (6), cispadire (7), and ondansetron (8).

We report a case of CBS successfully treated with mirtazapine.

Case Report

An African-American man, aged 59 years, was admitted to a tertiary care community hospital for pneumonia. The patient had a history of type 2 diabetes, chronic renal failure requiring dialysis, and bilateral macular degeneration. His visual acuity was almost “hand motion.” On the fifth day of his hospitalization, psychiatry was consulted because
he was experiencing visual hallucinations. The patient had no psychiatric history and had never taken any psychotropics. To everyone’s astonishment, he had been visually hallucinating for at least 3 years and had never mentioned this to any one except his family members. His hallucinations consisted of seeing groups of people and farm animals, more frequently in the evening. They usually occurred during periods of wakefulness and with his eyes open. He reported them as nonthreatening and felt comfortable seeing very vivid pictures. He acknowledged some sleep problems and inconsistent appetite but gave no evidence of having depressive disorder or any other psychiatric condition. His cognition was intact, as he scored 27/30 on the Mini-Mental State Examination (3), only losing points on the visual items. A complete blood count and complete metabolic profile revealed results consistent with chronic renal failure. A magnetic resonance imaging study showed age-related atrophic changes. He was given the diagnosis of CBS, and we started him on mirtazapine 7.5 mg at bedtime. Supportive therapy and therapy to increase insight into his condition were also considered. His antibiotics were gradually weaned as his pneumonia slowly resolved. The patient’s sleep pattern improved, and his visual hallucinations remitted within the next 3 days. He had no recurrence of visual hallucinations after a month of follow-up with his primary care physician.

We chose mirtazapine for several reasons. First, cisapride and ondansetron are also 5-HT3 receptor antagonists (8) and have been shown to treat CBS. Mirtazapine is a presynaptic alpha 2 antagonist that acts by increasing noradrenergic and serotonergic neurotransmission (8). The enhancement of serotonergic neurotransmission is specifically mediated via 5-HT1 receptors, as mirtazapine is a postsynaptic serotonergic 5-HT2 and 5-HT3 antagonist. Second, mirtazapine has very weak muscarinic anticholinergic and antihistaminic properties and is routinely used by geriatric psychiatrists to treat elderly patients. Third, atypical antipsychotics are associated with several risks, including adverse metabolic effects. Although this patient was receiving diazoxide, he was not taking erythropoietin, which can itself cause visual hallucinations (10).

We believe that, because of its unique pharmacologic properties, mirtazapine is a safe, effective, and well-tolerated treatment option for CBS. Last, reassurance and physician awareness are also vital to successful management of this condition.

References

Case Report
Mrs A is white and aged 64 years. For the past decade, she has suffered from generalized anxiety disorder, dysthyemic disorder (diagnosed according to DSM-IV criteria), and pathological skin picking. She had been followed by her primary care physician for 2 years before being referred for a psychiatric evaluation. Prior to her psychiatric evaluation, she had received adequate trials of amitriptyline and sertraline; she had also received a low dosage of a benzodiazepine (lorazepam, 1 mg twice daily). She showed modest improvement in her anxiety and depressive symptoms; however, she continued to engage in skin picking throughout the day and noted that it constituted approximately 2 to 3 hours of her total waking time. At the time of her psychiatric evaluation, she noted that, unless a family member brought it to her attention, she was frequently unaware that she was excoriating her skin. Her scalp, the back of her neck, and her arms evidenced extensive and multiple sites of chronic excoriation that were a source of much embarrassment to her. She acknowledged that her skin picking was highly distressing but reiterated, “I don’t even know I’m doing it most of the time.” Behavioural strategies of self-monitoring and habit reversal did little to diminish the repetitive scratching activity.

To pharmacologically target the skin-picking behaviours, her sertraline was tapered and fluoxetine was initiated at 20 mg daily. Her anxiety and mood symptoms continued to be well controlled over a 12-week period, but the frequency of the interfering skin-picking behaviours was unaffected. Fluoxetine was increased to 40 mg daily and maintained at that dosage for an additional 4 weeks. The repetitive behaviours decreased slightly. Given olanzapine’s apparent effectiveness,

Olanzapine Augmentation of Fluoxetine in the Treatment of Pathological Skin Picking
Dear Editor: Pathological skin picking, also known as psychogenic excoriation, is characterized by excessive, stereotypic scratching or picking of normal skin or skin with minor irregularities; it leads to tissue damage and personal distress (1). Pathological skin picking is not given a formal diagnostic category in the DSM-IV, but it may be either a primary impulse-control disorder or a symptom of a mood, anxiety, or delusional disorder (2). Current literature suggests the possibility of a clinical and conceptual overlap with the impulse-control disorder trichotillomania (3). I describe the case of a geriatric patient whose pathological skin picking did not respond to various antidepressant agents, including serotonergic reuptake inhibitors, until a low dosage of olanzapine was added to her maintenance dosage of fluoxetine. Although there are case reports wherein olanzapine augmentation of fluoxetine decreased the repetitive behaviours of trichotillomania, this appears to be the first report of this combination’s dramatically improving pathological skin picking.

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References
when combined with fluoxetine, in controlling symptoms of trichotillomania (4,5), a trial of an olanzapine augmentation was performed. Olanzapine was initiated at 5 mg at night while the fluoxetine was maintained at 40 mg daily. After 2 weeks, Mrs A stated that the time given to skin picking had been reduced to about 30 minutes daily, and after 6 weeks, the behaviours were almost entirely extinguished, apart from occasional scratching of her forearm. She stated that she was much more aware of her impulse to pick at her skin and that she was able to exert a behavioural brake to the activity. She reported no significant change in her mood or anxiety symptoms since she began olanzapine augmentation. She also denied side effects to the combination treatment and continues to maintain a therapeutic response after 6 months.

In the literature, fluoxetine has been reported to be especially beneficial in reducing pathological skin-picking behaviours (6,7). With this particular patient, fluoxetine was quite effective in reducing mood and anxiety symptoms but of slight benefit in diminishing the psychogenic excoriation. To successfully treat trichotillomania, the literature clearly supports augmenting selective serotonin reuptake inhibitors, specifically fluoxetine, with olanzapine. Given the possible clinical similarities between trichotillomania and skin picking, as well as the antihistaminic and antimpulsive properties of olanzapine (8), clinicians may find this augmentation strategy useful in treating individuals who present with this difficult-to-treat condition. This case suggests a possible role for olanzapine augmentation of fluoxetine in the treatment of individuals with comorbid mood and anxiety disorders who present with refractory skin picking.

References


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Internet Use in Adolescents: Hobby or Avoidance

Dear Editor: We report on 2 cases of excessive Internet use in adolescents presenting for psychiatric consultation. Is Internet use a hobby, an avoidance of relational deficits, or a contemporary manifestation of psychiatric symptomatology such as obsessive-compulsive disorder? There is mounting concern about the effects of computer use on behaviour and on social and mental health, owing to the increasing amount of time children and adolescents spend using computers.

Unfortunately, there is limited research in this area; one study shows that girls who had high levels of conflict with parents or who were highly troubled were more likely than other girls to have close online relationships, as were boys who had low levels of communication with parents or were highly troubled, compared with other boys (1). Another study shows computer use is linked to slightly better academic performance. Although little evidence indicates that moderate use of computers to play games has a negative impact on children’s friendships and family relationships, recent survey data show that increased use of the Internet may be linked to increases in loneliness and depression (2). Although not formally recognized in the DSM-IV, some psychologists have assigned diagnostic criteria to Internet addiction disorder (IAD) that are very similar to the addictive model of tolerance, withdrawal, and impairment of social and occupational function (3,4). However, caution is needed in applying the concept of behavioural addiction to computer-related behaviour. Classifying individuals as exhibiting pathological computer use by using checklists based on adaptations of DSM criteria for pathological gambling is likely to overestimate the number of people addicted to computing activities (5).

Case Report 1

Tomas, aged 16 years, lives in a city with his middle-class professional parents. He was referred to the adolescent clinic for spending 16 hours daily using the Internet, for being absent from school, and for having lowered frustration tolerance and behaviour problems, such as stealing from home and charging costs to the home telephone. Tomas’ parents feel helpless in controlling their son’s behaviour. He has a history of impulsiveness, easy boredom, lacking capacity to postpone gratification, lacking close friendships, irritability and externalizing behaviours, and difficulty with coping skills. Psychometric testing reveals a full-scale IQ of 116, Thematic Apperception Test reveals themes of sadness and depression, isolation, and poor self-esteem.

Case Report 2

Leela, aged 14 years, was seen at her parents request after threatening to kill herself. The history reveals 6 months of ongoing family conflict owing to Leela’s refusal to attend school and to her spending all night using the Internet and refusing to sleep. She was not eating meals with the family. A crisis arose when Leela’s parents disconnected the Internet; she threatened to kill herself (2). Leela is a Grade 9 student referred for school refusal. She has had a difficult time in school, being teased, and has found it difficult to make and keep friends. She has been shy and anxious. Since getting the Internet connection, however, Leela believes that she has friends in the chat rooms who understand her feelings and that she is able to say whatever she likes without fear of repercussion; for once, she does not feel “worthless.” Leela admits that she is using the Internet to the exclusion of everything else, but she cannot stop. She is irrational and argumentative around the issue of Internet use.

Discussion

These cases illustrate 2 different aspects of excessive Internet use. Case 1 seems to fit the addictive model, whereas in Case 2, there is evidence of anxious-avoidant traits that are being handled through the Internet. In both cases, the parents are helpless in curbing the behaviours and look to psychiatry to provide intervention and advice. In both cases, treatment of the underlying psychopathology was advised. In Case 2, successful treatment of the
anxiety disorder with cognitive-behavioural therapy, social skills group, and parental behaviour interventions led to Internet use reduced to appropriate weekly hours and to school reintegration.

**References**


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**Light Therapy, Nonseasonal Depression, and Night Eating Syndrome**

*Dear Editor:* The prevalence of night eating syndrome (NES) (that is, morning anorexia, evening hyperphagia, and insomnia) in the general population (1.5%) is lower than in obese patients (8.9% to 27%) (1) and is greater among men (2). We recently suggested that bright light therapy may cure NES and reduce body weight in obese patients (3). We report on the first case of a non-obese man suffering from NES and treated with light therapy.

A nonobese man (that is, Body Mass Index 23) aged 46 years presented as an outpatient for poor sleep quality and fatigue for 4 months. A thorough psychiatric examination by a senior psychiatrist and a record of food consumption (that is, energy and macro-nutrient content) showed nonseasonal major depressive disorder with moderate recurrent episodes and partial remission between episodes according to DSM-IV criteria. The structured interview guide for the Hamilton Depression Rating Scale-Seasonal Affective Disorder (HDRS-SAD) (that is, the usual 17 items of the HDRS plus 8 items assessing atypical symptoms) and the Hospital Anxiety and Depression (HAD) scale was used to assess severity. Initial scores were as follows: HDRS 17-item score 16, 8-item atypical score 20; HAD total score 16, HAD Depression subscore 8, and HAD Anxiety subscore 8. The patient also met the following provisional criteria for NES (4): morning anorexia; evening hyperphagia, in which at least 50% of daily energy intake is consumed after the last evening meal (this patient consumed 80% after 8:00 PM); awakenings at least once nightly (this patient awoke at 11:00 PM, 3:00 AM, and 5:00 AM nightly); and consumption of snacks during awakenings (70% carbohydrate-rich nighttime snacks with carbohydrate-to-protein ratio of 7:1 to restore patient’s disrupted sleep). These criteria persisted for at least 4 months.

After 14 consecutive morning sessions of light therapy (10 000 lux for 30 minutes), the patient no longer fulfilled the DSM-IV criteria for depression, and scores were significantly reduced: HDRS 17-item score 4, 8-item atypical score 3, total HAD score 5, HAD Depression subscore 3, and HAD Anxiety subscore 2. Further, NES criteria no longer remained.

**Discussion**

This case suggests that NES may be associated with depression in patients who are nonobese as well (5). Thus the NES and depressive symptoms appeared concomitantly, and both improved after exposure to light. Since bright light therapy in nonseasonal major depressive disorder yielded inconsistent results (6), we suggest that NES may be a predictor for its efficacy in depression.

**References**


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