Letters to the Editor

Safety of Clozapine in 2 Successive Pregnancies

Dear Editor: With the introduction of atypical antipsychotics, it was predicted that the incidence of hyperprolactinemia-induced infertility problems would decrease. However, the use of atypical antipsychotics during pregnancy is an area with more questions than answers. We present a case wherein clozapine therapy was continued successfully over 2 consecutive pregnancies.

Case Report

MK, aged 25 years and a housewife of rural background, was premorbidly well adjusted, with a family history of depression. She had an acute-onset 7-year continuous illness precipitated by the death of her father. The illness was characterized by auditory hallucinations (that is, commenting and commanding), tactile hallucinations, passivity phenomenon, aggression, negative symptoms, and disturbed biofunctions. There was no history suggestive of depression or mania, substance abuse, or head injury. During this period, treatment tolerance or resistance was seen with various antipsychotics (that is, chlorpromazine, haloperidol, trifluoperazine, and risperidone). Hence, clozapine was initiated after baseline investigations up to 400 mg daily. With 8 months of treatment, significant improvement in psychopathology and functioning occurred, with associated weight gain of 10 kg. Over the next 3 years, clozapine was gradually reduced to 200 mg daily with no reemergence of positive symptoms but persistence of negative symptoms. MK was married in early 2001 and continued to take clozapine. She was informed about the risk of congenital malformations with antipsychotics and was advised to use contraception. However, she conceived after 6 months of marriage and continued with the pregnancy while on clozapine 100 mg daily. PIH occurred at 30 weeks and was managed with methyldopate hydrochloride 1500 mg daily. At 39 weeks, a 2.8-kg girl in breech presentation was delivered by elective cesarean section. The baby’s Apgar scores were 7 and 9 at 1 and 5 minutes, respectively, with no postnatal complications. Developmental status was normal until age 6 months.

Discussion

Clozapine is a “Category B” drug with no controlled studies in pregnant women. Available data are generally in the form of case reports or series (1).

A recent review reported 5 congenital malformations and 5 perinatal syndromes in each of 61 children exposed to clozapine (1). Various associations with maternal exposure include floppy infant syndrome (2), neonatal seizures (3), new onset or worsening of gestational diabetes mellitus with shoulder dystocia (4,5), decreased variability of fetal heart rate (6), and intrauterine growth retardation with oligohydramnios (7). However, use of drugs concomitant with malnutrition and family history of diabetes mellitus have been certain confounding factors.

Conversely, Waldman and others reported at least 15 normal births with maternal clozapine exposure and have discounted definite association between clozapine and congenital malformations (4).

In the case of our patient, clozapine was not associated with any adverse effects on the fetus (including congenital malformations) over 2 consecutive pregnancies, which adds to clozapine’s safety data and is in keeping with a recent suggestion that it may be justifiable to continue clozapine during pregnancy when benefits outweigh the risks (8).

References


Nitin Gupta, MD, Staffordshire, UK; Sandeep Grover, MD, Chandigarh, India

Revisiting the Diagnostic Challenges of Secondary Mania and Bipolar Disorder in a Patient With Borderline Hyperthyroidism

Dear Editor: It can be a challenge to differentiate between a manic episode in bipolar disorder (BD) and a manic episode owing to a general medical condition. There are few case reports of BD associated with hyperthyroidism, despite the fact that symptoms of a manic episode overlap with those of hyperthyroidism (1–6). This may be attributed to problems in establishing etiology because of past psychiatric history and previous treatment with lithium, which can have antithyroid effects (7). There are few studies in the literature carried out with lithium-naïve patients regarding thyroid dysfunction and BD (8), and the role of a borderline hyperthyroid state in patients is not well described. Therefore, we report on a drug-naïve patient who presented with a manic episode and was found to have borderline hyperthyroidism.

Case Report

A woman, aged 54 years, presented at a university hospital with a 3-week history of elevated mood, grandiosity, incessant activity, and decreased sleep. There was no psychiatric history, and she had otherwise been functioning well. Her medical and family histories were unremarkable, and she was not on any medications. Her mental status exam revealed pressured speech and flight of ideas. On admission, her thyroid profile revealed the following: thyroid-stimulating hormone...
(TSH) 0.11 mU/L (0.35 to 5.5), free triiodothyronine (T3) 5.2 (3.5 to 6.5), free thyroxine (T4) 18 (11 to 23), and thyroglobulin 25.5 (< 43). Other routine blood tests and computed tomography imaging of her head were normal. Given the suppressed TSH, she was diagnosed with borderline hyperthyroidism.

While in hospital, the patient received olanzapine dissolvable 5 to 7.5 mg once daily, and her manic symptoms abated over a week-long hospital stay. After discharge, she did not continue taking medications. On follow-up a month later, the patient had resumed her usual activities and was euthymic. At this time, lab tests were done and showed a normal thyroid profile.

In the case of borderline hyperthyroidism, it may be difficult to establish whether the hyperthyroid state is the etiologic cause, a contributing factor to the manic episode, or simply comorbid with BD. In this case, it is reasonable to postulate that the hyperthyroid state contributed to the manic episode or precipitated a manic episode in a patient predisposed to BD. This is further supported by the fact that the patient’s symptoms resolved without further medications and were coincident with normalization of her thyroid profile. This reinforces the concept that perturbations in the thyroid function may be tied to manic symptomatology. At this time, the patient’s preliminary diagnosis is secondary mania, and longitudinal follow-up of her mood symptoms and thyroid function may lead to a more definitive diagnosis. If she had another manic episode without clinical or laboratory indicators of hyperthyroidism, particularly elevated triglycerides, they have been identified (3). Dyslipidaemia is a major contributor to cardiovascular disease and 36% of deaths (over 78 900) in Canada yearly are due to diseases of the circulatory system (4). Raised C-reactive protein is also seen as a strong predictor of a cardiovascular event (5).

Nine months ago, I began routinely testing patients’ lipid, C-reactive protein, and blood glucose levels. So far, I have tested 89 patients, about one-sixth of my practice. Among those patients, 67% had a two-fold or more risk of a cardiovascular event, based on a raised total cholesterol/HDL-C ratio. This held true whether the patient was on a conventional antipsychotic, (11 of 13 or 85%), an ATP (25 of 41 or 61%), or no antipsychotic (25 of 35 or 66%). Elevated C-reactive protein was present in 8% of patients. The screening process did not allow for any attribution of cause or determination of how long the elevated levels had been present.

Monitoring weight, blood glucose, and lipids should become routine psychiatric practice, and where necessary, the primary physician should be alerted. These measures are not routinely done in psychiatric inpatient settings, and a recent survey found that only about 50% of inpatients were checked for blood glucose and weight (6). Encouraging healthy diets, weight loss, and exercise is necessary. However, results of exercise programs may not be encouraging, as motivation is crucial (7). Medical management, including the use of statins, may be indicated.

If my results are characteristic of a general adult psychiatric population, all patients, regardless of diagnosis and treatment, should be appropriately screened and monitored. Psychiatric populations, for various reasons, appear to be at increased risk of cardiovascular disease.

References


Emiko Moniwa, Burnaby, British Columbia; T Warren Lee, MD, New Haven, Connecticut; Jodi Lofchy, MD, FRCP, Toronto, Ontario

Dyslipidaemia and Psychiatric Patients

Dear Editor: Associations between atypical antipsychotics (ATPs), weight gain (1), and diabetes (2) have been reported, and links between some ATPs and dyslipidaemia, particularly elevated triglycerides, have been identified (3). Dyslipidaemia is a major contributor to cardiovascular disease and 36% of deaths (over 78 900) in Canada yearly are due to diseases of the circulatory system (4). Raised C-reactive protein is also seen as a strong predictor of a cardiovascular event (5).

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If my results are characteristic of a general adult psychiatric population, all patients, regardless of diagnosis and treatment, should be appropriately screened and monitored. Psychiatric populations, for various reasons, appear to be at increased risk of cardiovascular disease.

References


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Dream Contents in Patients With Major Depressive Disorder

Dear Editor: The ancients attributed great importance to dreams. Special temples built by the ancient Chinese, Egyptians, Greeks, and Romans were sanctuaries to which people retreated to understand the meaning of their dreams. Hippocrates (469–399 BC) and Galen (ca 130 AD) believed dreams could have psychological and diagnostic utility (1). Dreaming is one of the interesting aspects of human beings, and physicians could find important information about their patients by exploring their dreams. Dreaming also has several psychological functions, one of which is mood regulation, in healthy subjects (2).
We observed that many of our patients with depression remembered repetitive dreams that were exhausting or frightening. In this case-controlled study, we examined the dream contents of 41 patients who met DSM-IV criteria for major depressive disorder (MDD) and 82 matched healthy subjects. We conducted the study at the Kerman psychiatric hospital in Iran. Subjects were requested to freely remember their recurrent dreams in the previous 2 months. Each dream that received at least a point 4 from Schredl’s dream questionnaire, was considered a recurrent dream (3). We organized dream contents into 14 main categories, using the Hall/Van de Castle major categories (4).

Results
The mean (SD) age of patients was 32.77 (7.83) years; 24 (58.54%) were women and 17 (46.41%) were men. Forty (97.6%) patients and 68 (82.9%) healthy subjects had at least one recurrent dream in the previous 2 months (odds ratio [OR] 68; 95%CI, 10.25 to 13.20; P = 0.002). In 40 (97.6%) patients and 10 (12.2%) control subjects, the recurrent dreams were frightening (OR 10, 95%CI, 1.33 to 208.92; P = 0.015).

Frequency distribution of dream content categories among patients with depression was as follows: death (87.80%), separation (70.73%), frightening animals (60.97%), frightening situations (58.53%), natural disasters (59.09%), aggression (48.78%), falling (43.90%), waste matters (43.90%), blood (41.49%), negative emotions (41.46%), bizarre elements (39.02%), punishment (26.83%), suicide and homicide (24.39%), and sexual harm (21.95%). All categories of dream content were more prevalent among patients with depression than among the control group.

Conclusion
In this study, we observed that many patients with depression whose chief complaint was not disturbing dreams had recurring and frightening dreams, when asked directly.

Exploring dream contents in patients with depression may be important from clinical points of view. For example, Agargun and others showed an association between repetitive and frightening dreams and suicidal tendency (5). Diminishing frequency or even disappearance of recurrent dreams in patients with MDD show the effectiveness of treatment with antidepressants (6). We recommend that physicians pay more attention to dream contents in patients with MDD, because these themes are unpleasant for patients, and trying to reduce them could help patients’ improvement.

References

Ali reza Ghaffari Nejad, MD; Rah yol Zahra Sanatinia, MD; Kiarash Yousof, MD, Kerman, Iran

Sensory Deprivation and Disorders of Perception

Dear Editor: Disorders of perceptions can be seen in schizophrenia, mania, delirium, metabolic and endocrinological disorders, epilepsy, brain tumors and drug intoxication, or by direct effects of some drugs (for example, hallucinogens). They can also be seen in the electrical stimulation of structures such as the amygdala and temporal cortex and in other pathologies of the brain (1). Perceptual disorders can also be observed, except for brain pathology or psychopathology. In these cases, it is largely suggested that the reason for perceptual disorders is sensory deprivation. Even though the mechanism is not well known, it is believed to be similar to phantom extremity phenomenon (2).

In the literature, there are articles about visual hallucinations observed in patients having visual defects without suffering from any psychiatric disorder (2–6). This was first claimed by Charles Bonnet, a Swiss scientist. Bonnet’s grandfather experienced live visual hallucinations at age 89 years, 11 years after having cataract surgery (3). Interestingly, Bonnet himself suffered from visual defects and afterward had similar symptoms. Morisier first named this Charles Bonnet Syndrome (CBS) in 1938 (4). Once the syndrome was named, many similar cases were reported, and diagnostic criteria were developed for CBS (5,6).

In this article, we report on the case of a patient with auditory hallucinations in the left ear only. The patient has suffered from hearing loss in the left ear for 5 years, but has not used a hearing aid and for the last 2 years.

Case Report
A woman, aged 63 years, complained that she was hearing voices for the past 1½ to 2 years and that the voices had increased in the last month, especially at night. At first, she was afraid of the voices and thought she was mad; then she realized they were not real. She had hearing loss in her left ear following a car accident 5 years ago, and it was recommended that she use a hearing aid. Since the aid disturbed her ear, she used it irregularly for 3 years and not at all thereafter. Five to 6 months later, she started to hear voices. Everything was normal in the patient’s psychiatric examination except for auditory hallucinations and anxious mood. There was no substance use or psychiatric illness in her history. Complete blood count, biochemical tests, thyroid function test, EEG, cranial computed tomography scan, and neurological examinations of the patient were normal. Her score on the Mini-Mental State Exam was 27. The Minnesota Multiphasic Personality Inventory and Beier Sentence Completion tests were evaluated as normal. Treatment was started with olanzapine 10 mg daily and diazepam 10 mg daily. Later, diazepam was stopped and continued by olanzapine 5 mg daily only. On the tenth day of treatment, the auditory hallucinations disappeared. The patient was observed for 10 months at regular intervals; she took olanzapine 5 mg daily and used a hearing aid for 7 months. During this time, no psychopathology had been detected.

How could sensory deprivation cause disorders of perception? Even though the mechanism is not well known, it is believed to work similarly to phantom extremity phenomenon. In phantom extremity phenomenon, the sensory deprivation could be due to amputation of a body part, damage of the sensory nerve, brachial plexus, or blockage of the spinal cord by anesthetic agent (2). In this case report, the sensory deprivation now caused by damage to the auditory ways after an accident. In both cases, the stimuli going to the cortex were blocked. However, how the perceptual disorders occur is still not known. Perhaps the hallucinations are due to receptor hypersensitivity. This could be explained by examining the effective mechanisms of hallucinogens and atypical antipsychotics. Hallucinogens and serotonin-dopamine antagonists (SDAs) act through the same receptors but create different effects. Hallucinogens activate 5HT2A, 5HT2C, and 5HT6.
receptors and cause hallucinations. In contrast, SDAs block these receptors and prevent the formation of hallucinations (9).

Could this patient be diagnosed with CBS? For now, the diagnosis criteria for CBS defined by various authors are as follows: The patient should have complex visual hallucinations; insight should be partially or totally protected (that is, hallucinations should be perceived as unreal); there should not be delirium, and there should not be hallucinations in the other sensory organs (5,6).

Our patient perceived the hallucinations as unreal and did not have delirium, but she could not be diagnosed with CBS because these criteria focus only on visual hallucinations. At this point the purpose is to possibly prevent discarding the diagnosis of not otherwise specified psychotic disorder, since auditory hallucinations are the most often observed perceptual disorders in schizophrenia. However, the patient’s psychiatric examination and psychometric evaluation proved she had no psychosis. Further, auditory hallucinations were only in the ear with hearing loss, and our patient perceived these as unreal. In the literature, CBS cases with auditory hallucinations and auditory hallucinations in deaf patients have rarely been mentioned (7,8,10,11). Thus, according to us, the diagnosis of this patient was CBS. Within this context, we suggest that the diagnostic criteria of CBS could be enhanced. Additionally, if it could be proven that the patient did not suffer from psychosis and other perceptual disorders, except from visual hallucinations that could be seen in the other sensory organs with sensory deprivation, the diagnosis could again be CBS.

References:


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Ankara, Turkey

Re: The Internet’s Impact on the Practice of Psychiatry

Dear Editor: Dr Styra recently reviewed the impact of the Internet on psychiatry (1). Described below is a novel example of the use of the Internet in providing care. Instant messaging services are one of the many uses of the Internet and are a popular method of communication among many Internet users.

Case Report

A young man with hearing impairment presented to the emergency department with his mother. He was referred for suicidal ideation at approximately 3:00 AM. To complete the psychiatric evaluation and to ensure confidentiality, attempts were made to obtain a sign language interpreter. An interpreter would be available only in the morning, and the wait was unacceptable to the patient. Attempts to use paper and pencil to complete the assessment were less than ideal. A novel idea of using an Internet instant messaging system to complete the assessment was offered. The patient used 2 computers that were already set up side by side to complete the assessment. The patient and clinician logged on and connected via the messenger service. The close approximation of the communication was complete with its own syntax and subtleties. The assessment was not ideal, it sufficed under the circumstances. The use of video will improve communication via instant messaging services. The cost of this service is considerably less than those associated with telepsychiatry, and it may be a useful tool in psychiatry in the future.

References


Krishna Balachandra, MD
London, Ontario

Response: The Internet’s Impact on the Practice of Psychiatry

Instant messaging (IM), also known as “chat,” is the text version of a phone call. It has gained rapid acceptance with teenagers and young adults, who prefer this form of communication over e-mail and phone calls, because of its immediacy and streamlined efficiency. It is similar to having a conversation with a person in the same room.

IM is gaining acceptance in the workplace and will likely become a core technology, much as e-mail is today. Security and auditing issues are critical concerns that must be addressed with respect to using IM in a health care environment, as patient confidentiality is of paramount importance. Security is a significant factor, since the text in an instant message is relayed to a Web server while en route to the person who will be the recipient of the message. This message can be viewed by anyone on the connection (that is, a service provider, employee, hacker, and so on). There are third party vendors that offer solutions to fill in the security and auditing gaps within IM by employing encryption algorithms and other measures.

Technical issues aside, Dr Balachandra’s example illustrates how IM technology can be used to improve patient service when human resources, such as an interpreter, are unavailable. IM is a technology that is familiar to the hearing-impaired community (1). IM also takes the guesswork out of penmanship as the message is in type and can easily be read. Dr Balachandra’s example is a novel one and is relevant to the psychiatric community. Research is required to determine best practices and the economics of this technique.

References


Rima Styra, MD, MEd, FRCPC
Toronto, Ontario
Dear Editor: We recently encountered the case of a woman who died in an atypical way as a result of coping with a breast mass by denial and avoidance. The patient, aged 48 years and white, lived at home with her husband and 2 young children. For 4 to 6 weeks preceding her death, she isolated herself from her family in the basement, explaining that she had personal issues that she was handling alone. She continued to work at her job, where her coworkers noted that she had become withdrawn. She was later found dead in the basement, surrounded by blood-soaked, foul-smelling clothing and gauze. There was a penetrating injury of the right breast and chest wall. An autopsy revealed that the injury was from a large ulcerated breast carcinoma, which eroded the chest wall and opened a large artery. There was no evidence of metastasis, which is the most common cause of death from breast cancer (1,2). Death owing to hemorrhage occurs in only approximately 9% of breast cancer patients (1,2) and is usually a complication of progressive disease. Death from a localized and usually curable primary tumor such as this one is most unusual.

We believe the patient’s denial and avoidance of her disease were psychosocial factors leading to this atypical mechanism of death from breast cancer. Denial can impede a woman’s willingness to undergo screening examination (3). A substantial number of women may not recognize a breast abnormality on their own, before it is found by screening (4,5). They may use avoidant coping mechanisms that prevent them from checking for potential disease (4). Women who experience denial prior to disease identification are also more likely to maintain that denial later in the course of disease (5).

After a diagnosis of breast cancer, a woman may experience denial based on fear of changes in self-image or sexuality or on concerns about the effect of the disease on her relationships with her partner or children (6). Coping strategies after diagnosis tend to assume a proactive character (3,7,8), however, and may include such activities as acceptance, positive reframing, use of religion, expressing emotion, adopting a fighting spirit, seeking support, asserting self-control, or diverting energy to other matters (7–9).

While proactive coping strategies may seem to caregivers to be more productive in dealing with a serious disease, denial is not inherently a poor coping strategy. It can provide relief of psychological distress during difficult periods of treatment (3,7) and may be associated with remaining recurrence-free or experiencing prolonged duration of survival in women with local disease (3,10,11). Denial does not necessarily lead to a mood disturbance or to poor adjustment to treatment (9). However, relying on denial as a significant coping strategy has disadvantages. Women who use denial or avoidance tend to experience more distress and poorer adjustment than do women who use proactive coping strategies (7,8,12). Of course, as this patient illustrates, denial and avoidance may impart a risk of missing a diagnosis and foregoing potentially lifesaving treatment.

References

Henry J Carson, MD; Richard Fiester, MD
Cedar Rapids, Iowa

Interferon-Induced Mania

Dear Editor: Hepatitis C is a chronic viral illness and a leading cause of cirrhosis and liver failure. Treatment of this condition is limited, with pegylated interferon-alpha (IFN-alpha) and antiviral drugs being the mainstay of treatment. Unfortunately, these drugs may have various side effects and a broad range of neuropsychiatric manifestations. We report on a case that illustrates mania secondary to antiviral agents.

Case Report

Mr A, a white man aged 41 years, was brought to the emergency room (ER) by his girlfriend when she noted that he had not been sleeping well and that he had been getting increasingly disorganized over the preceding week. It appears that he had been started on IFN-alpha after a diagnosis of hepatitis C was made by his gastroenterologist. He began to exhibit mood instability and subsequently presented to the ER with a several-day episode of poor sleep, along with delusions of grandiosity, persecution, and reference. His thoughts were disorganized, and he was belligerent. His girlfriend reported that he was more religiously preoccupied than usual. There was no evidence of intent to harm himself or others. He did not have a psychiatric history prior to starting interferon treatment.

Mr A was admitted to hospital and, after consulting with his gastroenterologist, interferon and ribavirin were withheld, and he was stabilized with haloperidol. He showed a remarkably swift recovery over the next few days, after which he was discharged home.

Discussion

Treatment of chronic hepatitis C is difficult and is further complicated by the fact that IFN-alpha is associated with severe neuropsychiatric adverse events in some patients. The presence of preexisting psychiatric diagnosis has not been shown to be a risk indicator (1). Presence of a subclinical neurological condition, however, may place the patient at a higher risk (2). In our patient, the timeline between drug administration and appearance of symptoms strongly implicates the drug as the cause. Since the psychopathophysiology of mania in IFN-alpha treatment is largely speculative, clinical risk stratification remains largely unsatisfactory. Controlled trials are needed to determine optimum treatment options in these complicated patients.

Funding and Support

None of the authors have any financial or professional connection to any product mentioned above.
The levetiracetam dosage was reduced to 1500 mg, and lamotrigine and primidone were continued unchanged. The following day intake of olanzapine 10 mg was started. Restraint was necessary for the first 2 days. On the evening of the second day, she calmed down and regained orientation and insight into her illness. On the third day, she reported acoustic hallucinations again: a female voice told her she had been given the wrong medicine. On the fourth day, her overall behaviour returned to normal, and she was readmitted to the neurological department.

In the literature, prevalence rates of levetiracetam-induced psychosis range from less than 1% to 1.4% (3–5). Mula and others report certain risk factors, including a history of febrile convulsions, status epilepticus or previous psychiatric history, or lamotrigine cotherapy (5). Only lamotrigine cotherapy was given in our patient. Levetiracetam-induced psychosis normally occurs about 1 week after the start of treatment. In our case, the patient received a stable dosage of levetiracetam for about 1 year, with psychosis occurring 1 week after the dosage was increased. However, psychosis is a side effect of levetiracetam therapy that can also occur after long-term treatment. In certain patients, risk-factors should be clinically monitored, specifically when the dosage is changed.

References

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Priapism
Dear Editor: Priapism is a rare but serious side effect of antipsychotic medication. Cases of ischemic priapism have been reported with most atypical antipsychotics (ATPs) (1). Priapism, which has both ischemic and high-flow subtypes, can also be associated with hematologic and vascular abnormalities, as well as spinal cord injury, neoplasms, and prostatitis. We present the case of a man who developed ischemic priapism on 3 occasions, while on 3 different ATPs.

Mr A is aged 44 years with a 23-year history of schizophrenia. He was taking risperidone 4 mg orally twice daily and trazodone 150 mg orally at bedtime when he first developed priapism. He received saline and phenylephrine cavernosal irrigation in emergency after having the priapism for 7 hours. We changed his treatment to quetiapine up to 600 mg daily. He developed ischemic priapism 24 days after starting quetiapine. Again, he required cavernosal irrigation to resolve a 10-hour period of priapism. We then treated him with olanzapine up to 20 mg daily. He developed ischemic priapism 53 days after starting olanzapine. This episode responded to phenylephrine 60 mg orally. Following this episode, we discovered that his fasting blood sugars were consistently greater than 15 mmol/L. His hemoglobin was 120 g/L. During the previous 2 years, his fasting blood sugars had ranged from 4.6 to 8.5 mmol/L. Further workup, including a CT scan of his pelvis, was negative. He was started on insulin andloxapine, and his fasting blood sugars normalized. We choseloxapine because it has minimal alpha 1 adrenoceptor blockade (2). He had no further episodes of priapism.

The patient had previously been on risperidone up to 8 mg daily with trazodone 50 to 100 mg daily for 2 years. He then had a 4-month trial of quetiapine up to 700 mg daily with trazodone 50 to 100 mg daily. He had no documented episodes of priapism during this period. His blood sugars were normal at that time. This case illustrates the potential risk of ischemic priapism from a range of ATPs aggravated by diabetes. The capacity for each of the pharmacologic agents to induce alpha 1 adrenoceptor blockade is likely the common denominator for the drug-induced priapism. All ATPs block alpha 1 adrenoceptors (3). Olanzapine has little action on alpha 2 adrenoceptors (3). Blockade of the sympathetic nervous system prevents its normal inhibitory influence on corpus cavernosal smooth muscle and on penile vascular inflow resistance. Both are required in mediating detumescence or maintaining a flaccid state. The high blood sugars likely facilitated the development of priapism by altering the normal status of the sympathetic nervous system systemically.
An Ounce of Prevention: “COPEing With Toddler Behaviour”

Dear Editor: Disruptive behaviour disorders are major mental health problems that are difficult to treat and costly in terms of suffering, violence, and damaged property. The most common reason for referral to children’s mental health services involves disruptive behaviours, that are quite stable from toddlerhood. Intervention may be more effective with younger children, who tend to have less severe behaviour problems; however, there are no evidence-based interventions specifically targeting toddlers (1). Group-based parent training can be effective, cost-efficient, and accessible, so we developed COPEing With Toddler Behaviour to train groups of parents in effective parenting strategies for toddlers (aged 12 to 36 months), using an active learning model (that is, COPE Community Parent Education, 2) that has been used successfully with other age groups. In the development phase of this community-based education group, we focused on enlisting and retaining parents of at-risk toddlers, completing evaluations, identifying any negative impact of the groups, and developing the content in response to feedback.

We successfully achieved our goals in developing the COPEing With Toddler Behaviour course (that is, enlisting and retaining parents of at-risk toddlers, completing evaluations, identifying any negative impact, and further developing the content). However, the pilot study involved a short version of the course, a small sample, no control group, and parent-report measures only. A randomized clinical trial represents the next step in this research.

Funding and Support
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Acknowledgements
I am grateful to Joe Ducharme and to the staff of the Infant–Parent Program who contributed to the development of this new service; to Leah Hatton, Catherine Hutchinson, Shawn McFadden, Michelle Mitchell, and Christina Ricciuti for their help with the pilot study; and to Harriet MacMillan, Michelle Mitchell, Mark Sanford, Louis Schmidt, and Larry Tuff for their helpful comments on earlier drafts of the manuscript.

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anxiety and escitalopram 10 mg daily for his depression was recommended. He was also referred for individual and group therapy. However, within a month, he stopped psychotherapy and gabapentin but continued the escitalopram. His dosage was increased to 30 mg daily over 3 months. This improved his depression and reduced his urges for on-line gaming. He erased the Everquest program from his computer and resumed his job and educational training. Follow-up testing showed a an ASI for Internet addiction decreased to 3, while the HDRS decreased to 5.

Current data on Internet gaming addiction suggests that on-line fantasy gaming is associated with introversion, low empathy, low self-esteem, (2) depression, and social isolation (3). On-line games allow players to assume a false identity in a virtual world and to interact freely with strangers, which provides an escape from reality. Treatment of these individuals should target comorbid psychiatric symptoms and their addiction to on-line gaming. In the absence of established guidelines, escitalopram, a selective serotonin reuptake inhibitor that is an effective treatment for depression, may provide additional benefit for patients with Internet gaming addiction.

**Funding and Support**
There was no external funding received for this study. There are no conflicts of interest.

**References**

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**ERRATUM**
In the letters to the editor of the November 2005 Canadian Journal of Psychiatry, Dr. Nasreen Roberts was incorrectly listed as Nasreen Roberts, PhD. The correct listing is Nasreen Roberts, MD, FRCPC. The Journal regrets this error.