Letters to the Editor

Re: Atypical Antipsychotic Use in Treating Adolescents and Young Adults with Developmental Disabilities

Dear Editor:

We work in an interdisciplinary community mental health service for persons with developmental disabilities (DDs) across the lifespan, and we therefore read with particular interest the article by Friedlander and others (1) on the use of atypical antipsychotics to treat adolescents and young adults with DDs.

In our clinical practice, we have also observed that some persons with DD at times experience a “dramatic and long-lasting response to low-dose atypical neuroleptics” but that “this population, however, seems particularly sensitive to neuroleptic-induced movement disorders (NIMDs), hence caution and close monitoring are required” (1). What we feel deserves greater emphasis for practitioners less familiar with this etiologically and symptomatically diverse population with different types and degrees of DD is that the diagnostic complexities involved are substantial. Even establishing a diagnosis of schizophrenia or autism–pervasive developmental disorder (PDD) can be challenging; differentiating symptoms of psychosis from symptoms of anxiety in persons with autism–PDD can be still more problematic. Friedlander and others did not comment on these challenges in their paper, nor did they attempt to tease apart whether outcome was due to the antipsychotic medication per se or whether and to what extent associated interventions with other medications or nonpharmacologic therapies may have contributed (a difficult thing to establish in a small sample). To identify those for whom atypical antipsychotics are effective, and in what context, further research on larger series is warranted.

Friedlander and others note that, even with their clinic’s conservative prescription practices, one-half the individuals in the sample were taking atypical antipsychotics—even when psychotic symptoms were not documented. We agree with the authors that, in the absence of clearly identified psychiatric disorders for which these medications are indicated, the practice of using either typical or atypical antipsychotics to treat behaviour disturbances is no longer tenable. We urge psychiatrists to identify and carefully monitor, in both their research and clinical practice, the target symptoms that the antipsychotic is intended to address, particularly when the symptoms are not psychotic (as may have been the case for many of the individuals with autism–PDD in the present sample). Proceding in this way helps to ensure that the old practice of overprescribing neuroleptics, noted by Friedlander and others, does not transfer into overprescribing newer antipsychotic medications, particularly in situations where the prescribing physician does not have access to a comprehensive multidisciplinary evaluation process.

The study by Friedlander and others is an important step toward evaluating the use of atypical antipsychotics in individuals with DDs, but the methodological and other concerns outlined above make it premature to endorse their use. It is our belief, supported by recent consensus guidelines (2–4), that a comprehensive evaluation to better understand the underlying cause of the symptoms and behaviour disturbances is essential—in particular, to ascertain whether these are indeed caused by a psychiatric disorder. This approach leads to more effective treatment, more appropriate and targeted use of antipsychotic medication, and fewer side effects.

References


Reply: Atypical Antipsychotic Use in Treating Adolescents and Young Adults With Developmental Disabilities

Dear Editor:

Thank you for giving us an opportunity to respond to the views of Bradley and colleagues. We are pleased that they highlighted the complex diagnostic issues involved in diagnosing Axis I mental disorders in individuals with developmental disorders (DDs). Prior to arriving at a diagnosis, all behavioural disorders in this population should be investigated carefully to see whether they represent non-verbal, indirect communications of distress caused typically by pain or psychosocial stress.

Our mental health support teams provide a full multidisciplinary evaluation, and all patients in our sample had behavioural–psychosocial interventions in addition to psychopharmacological
treatment. We intended our paper to illustrate the widespread use of atypical antipsychotic treatment in clinically referred youth with DDs and behavioural problems. How much improvement can be attributed to the atypical antipsychotics is a complex question that indicates the urgent need for more double-blind placebo-controlled trials in youth with DDs.

Our study highlights the need for great caution when using these medications in this population and notes atypical neuroleptic–induced movement disorders not previously well documented.

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Evidence Supports Validity of Seasonal Affective Disorder

Dear Editor:

We were surprised to read in a recent editorial that Dr Paul Grof questions the validity of the diagnosis of seasonal affective disorder (SAD). He writes that “the evidence for the existence of SAD itself is still missing, more than 20 years after it was proposed. In clinical course, genetic, and treatment studies, there is still no convincing justification for the diagnosis of an independent seasonal illness” (1, p 124). This statement seems based more on idiosyncratic opinion than on scientific evidence. First, it is worth clarifying that the DSM-IV classifies SAD as a seasonal pattern specifier for major depressive disorder (MDD) (2), and hence, it is regarded as a subtype of depression rather than an independent diagnostic category. Second, there is ample evidence to demonstrate that seasonal pattern is a valid specifier for MDD and that light therapy is an effective treatment for SAD. Since the condition was first described in 1984, nearly 1000 articles have been indexed on Medline under the heading, “seasonal affective disorder.” Indeed, extensive reviews support an argument that there is more evidence for the validity of SAD than for many other well-recognised depressive subtypes, including bipolar II disorder, atypical depression, and postpartum depression (3,4). SAD and its treatment have also been comprehensively reviewed and included in Canadian (5), American (6), and international (7) evidence-based guidelines for the treatment of depressive disorders. We suggest that it is time to leave behind the argument of whether SAD is a valid diagnosis and concentrate instead upon determining the pathophysiology and most effective methods of identifying and treating this prevalent and disabling subtype of depression.

References

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Reply: Evidence Supports Validity of Seasonal Affective Disorder

Dear Editor:

Let me thank Dr Michalak and Dr Lam for bringing to our attention an issue of major clinical importance—seasonal affective disorder (SAD). To illustrate the potential significance of such a disorder: if it were proven to recur consistently in the same season it could be treated intermittently, without exposing patients to the well-known adverse effects of chronic pharmacotherapy.

Dr Michalak and Dr Lam express surprise that I do not share their belief in SAD as a distinct subtype. The reason is simple, as I mentioned in my editorial: “In clinical course, genetic, and treatment studies, there is still no convincing justification” (1, p 124) for such an entity—and I am in good company when I conclude that SAD remains an elusive fiction (2,3).

A striking seasonality of episode onsets certainly exists for groups of patients with mood disorders, but individuals who experience recurrences in the same season for a few years lose this pattern later. The database from an international study of 1309 patients, developed and published before the present enchantment with SAD, shows that individual patients do not experience recurrences in the same season more often than might be expected by chance (4). Further, there is not a single patient in the study with recurrences continuing in the same season over the lifetime. Studies demonstrating that patients who initially meet the SAD criteria continue meeting them over time, are also missing in the literature.

To defend their belief, Dr Michalak and Dr Lam refer to what they consider to be indisputable authorities: the DSM-IV (5) and the International Guidelines (6). However, the DSM-IV committees refused to recognize SAD as an entity,
politely downgrading it to a simple “qualifier.” When I questioned Dr M Bauer, chair of the group that produced the International Guidelines, about the 2-paragraph statement on SAD, he wrote that the statement was based exclusively on “a review of literature, not on investigation.” Interestingly, the Guidelines’ authoritative literature turns out to be a couple of papers by none other than Dr Lam himself.

I agree with Dr Michalak and Dr Lam on 2 points. First, I agree that light therapy works—but it is similarly helpful in nonseasonal mood disorders (7), and some medications, such as tranylcypromine, work even better in reputed cases of SAD. Second, I agree that a large number of articles have indeed been published about SAD—but frequent repetition alone does not make the disorder real. In fact, that’s how myths have often been created in psychiatry: consider, for example, the extensive earlier literature on entities such as “involutional melancholia” and “anniversary depression” (the psychoanalytic precursor of SAD). Without evidence, such entities sooner or later become history.

I have collected lifetime data on the clinical course of nearly 2000 patients with mood disorders, and I have approached 2 colleagues who claim to specialize in SAD, yet I still have not come across a single patient with several recurrences limited mostly to a particular season. If Dr Michalak and Dr Lam have at least a couple of patients who actually continue meeting the criteria for SAD for an extended period, I would be happy to interview the patients and publicly recant my solid skepticism about SAD.

References


Paul Grof, MD, FRCPC
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Seasonal Affective Disorder:
The Latitude Hypothesis Revisited

Dear Editor:

Axelsson and others’ recent article concerning the prevalence of seasonal affective disorder (SAD) in people of Icelandic decent makes for interesting reading (1). The authors report that people of wholly Icelandic decent living in Winnipeg, Manitoba, show significantly lower rates of seasonality than do residents of non-Icelandic decent (as measured by the Seasonal Pattern Assessment Questionnaire [SPAQ] [2]). This finding complements those of an earlier study conducted in the Interlake District of Manitoba (3). The authors conclude that genetic factors play an important role in the etiology of SAD, and they express some doubt regarding the veracity of the “latitude hypothesis,” which posits a higher prevalence of SAD in more northern latitudes, owing to the shorter winter day.

An earlier review of epidemiological studies in this area did not find a significant overall correlation between prevalence of SAD and latitude ($r_{n-13} = 0.07$, $P = 0.42$) (4). However, the authors did observe meaningful correlations between SAD and latitude when the studies were split into those conducted in North America ($r_{n-7} = 0.90$, $P = 0.003$) and those conducted in Europe ($r_{n-6} = 0.70$, $P = 0.06$). While this review provided a useful contribution, it had some limitations: it included several studies not published in peer-reviewed journals, and it omitted published work conducted in areas outside North America and Europe.

To further inform this issue, we performed a slightly different quantitative synthesis of the literature. We included all peer-reviewed studies published before October 2000 that examined the prevalence of SAD using the SPAQ ($n = 22$), irrespective of the country in which they were conducted. We analyzed the studies in 2 separate categories: those conducted in general population samples (which are more representative of overall prevalence at any given latitude) and those performed in specific subpopulations. Where studies had been performed over several latitudes, we calculated mean prevalence rates and latitudes.

The results of this analysis showed a significant correlation between SAD and latitude in the general population studies ($r_{n-12} = 0.66$, $P = 0.019$) and an insignificant correlation in the specific subgroup studies ($r_{n-10} = 0.34$, $P = 0.34$).

Our results suggest that the prevalence of SAD in the general population does increase with higher latitude, consistent with the latitude hypothesis. However, although latitude does reflect the daily photoperiod in a given location, it is likely to be only a crude measure of other variables, such as climatic conditions. These studies are also limited by the fact that the SPAQ does not provide clinical diagnoses. We agree with Axelsson and others’ view (1) that SAD probably has a complex etiology and pathophysiology (5) influenced by several variables, such as environment, genetics, sociocultural context, and psychosocial factors. Nevertheless, given the study results, we do not wish to potentially “throw the baby out with the bathwater” and discontinue studying the relation between SAD and latitude.
Dear Editor:

Posttraumatic stress disorder (PTSD) can be a debilitating condition with impairment as bad as, or worse than, that caused by other psychiatric illnesses (1). Symptomatology includes a triad of symptoms from 3 clusters: persistent re-experiencing of the trauma (for example, intrusive and distressing recollections, nightmares, and dissociative flashbacks); persistent avoidance of stimuli associated with the trauma; and persistent symptoms of increased autonomic arousal (2). Because the symptoms of PTSD can vary across the symptom clusters, medications from different classes are needed to treat patients. Based on the hypothesis that kindling of the limbic nuclei occurs following the trauma (3–5), anticonvulsants such as carbamazepine and valproate were used to treat PTSD as far back as the mid-1980s to the early 1990s, (6,7). Newer anticonvulsants such as lamotrigine, gabapentin, and topiramate have also been used (8–10).

I present the case of a patient diagnosed with PTSD and major depressive disorder (MDD) who had taken multiple medications in the past and received benefit from tiagabine. To date, there are no reports in the literature describing the use of tiagabine to treat PTSD.

Case Report

Mr A is a 43-year-old man with a history of PTSD and MDD. His illness caused significant impairment over the years, and he had been treated with the following agents: sertraline, paroxetine, fluoxetine, bupropion augmented with lithium, trazadone, nefazodone, gabapentin, and topiramate.

Upon consultation, Mr A agreed to a trial of venlafaxine extended release (XR), started at 37.5 mg daily and titrated to 225 mg daily over 3 weeks. After 1 month, he noted an improvement in his depressive symptoms, but he was still experiencing intrusive thoughts, nightmares, and hypervigilance. He agreed to a trial of tiagabine, initiated at 2 mg daily. His dosage was increased by weekly 2 mg increments to 8 mg daily, at which level he began to notice an improvement in his intrusive thoughts and nightmares—even through the traumatic events of September 11, 2001, which provided a flood of external cues. He occasionally takes zolpidem tartrate 10 mg nightly for insomnia, but over the past 6 months he has improved significantly, to the point of returning to work full-time. He has improved significantly, to the point of returning to work full-time. He cannot recall feeling this stable during the last 10 years.

Tiagabine is FDA-approved as an adjunct anticonvulsant for the treatment of partial seizures (11). It is thought to exert its action by inhibiting GABA from the synaptic cleft (12,13). Tiagabine has also been shown to have antikindling potency (14), making it a potential candidate in treating PTSD. This patient’s improvement might have been due to a combination of agents, but the addition of tiagabine clearly led to a reduction in the reexperiencing symptoms. Further controlled studies are needed to investigate the efficacy of tiagabine in treating PTSD.

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Assessing Pain Tolerance in a Patient With Acute Psychosis

Dear Editor:

Increased pain tolerance in patients with psychosis has been recognized in the literature (1,2), but few studies have determined the etiology of this phenomenon. Rather, they present conflicting evidence to etiologic hypotheses. Can J Psychiatry 2002;47:153–8.


evidence suggesting psychological (3), biological (4), and pharmacologic (5) explanations. Regardless of its etiology, however, the careful assessment of how pain tolerance interacts with delusional ideation in patients recently hospitalized during an acute psychotic episode cannot be stressed enough. Simply diagnosing and assessing for psychotic symptomatology is not adequate. Failure to adequately assess the intricacies of a patient’s delusion system and pain sensitivity can result in serious medical consequences. The following case report will illustrate this important matter.

Case Report

Mr A is a 38-year-old Haitian-American with a 16-year history of schizoaffective disorder. He was admitted to an inpatient psychiatric hospital during his most recent psychotic episode. When admitted, Mr A was experiencing auditory command hallucinations and suicidal ideation. His command hallucinations were instructing him to commit suicide by overdosing on his prescribed neuroleptic (haloperidol) while consuming alcohol. He denied previous substance-abuse issues. Fifteen years earlier, he had followed command hallucinations to self-amputate his penis as a punishment for not yet having had sexual intercourse. During his most recent hospitalization, he complained to the nursing staff about problems with urination. (Although difficulties with urination can occur as a result of penile amputation, he had not reported any prior difficulty.) When evaluated after this complaint, the patient reported that his problems with urination had ceased and that he was able to urinate adequately without difficulty. However, although he initially denied any problems, a careful inquiry revealed that he was also experiencing mild abdominal pain which had commenced after he had experienced auditory hallucinations a few hours earlier. During this inquiry, the patient admitted to additional command hallucinations instructing him to “make [his testicles] numb.” He reported that the auditory hallucinations instructed him to squeeze his testicles. Since squeezing did not provide adequate numbness, he subsequently tied the top of his scrotum with a shoelace, which he had not yet untied. A physical examination revealed a shoelace tied around the apical part of the scrotum. The shoelace was then subsequently cut. A sonogram revealed a hydrocele of the testicles, which might eventually have led to vascular necrosis.

Discussion

In this case report, the patient’s ability to minimize his experience of pain and the consequences of his behaviour could have led to severe medical repercussions. During an acute psychotic episode, patients can experience significantly increased pain thresholds, a factor that should be considered as part of a complete assessment of delusional ideation. In this case, failure to thoroughly assess the extent of this patient’s delusion and pain threshold might have resulted in the loss of his testicles. Further research investigating the etiology and epidemiology of increased pain tolerance in patients with psychosis will improve assessments in this area and may also help to prevent serious medical consequences.

References


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Musical Hallucinations During a Treatment With Benzodiazepine

Dear Editor:

Musical hallucinations have been associated with deafness (1), epilepsy (2), organic brain disease (3), schizophrenia (4), and drugs (for example, propranolol [5] or salicylates [6]). We report the case of a middle-aged woman who presented musical hallucinations after starting a benzodiazepine treatment.

Case Report

A professional woman, aged 45 years, suffered from moderate depression with anxiety following an overload of work. The first manifestations were fatigue, a functional intestinal disorder, and a sleep disorder. After a couple of weeks, lormetazepam, a benzodiazepine available in Europe and Australia, was introduced at a dosage of 4 mg daily. After a few days, the patient noticed the appearance of musical auditory hallucinations like children’s songs. The intensity of these hallucinations decreased when she was concentrating on a task or had a conversation. The musical hallucinations persisted for 4 months, at which time their characteristics changed when amitriptyline was introduced and rapidly increased to 150 mg daily while the dosage of lormetazepam was decreased from 4 mg daily to 2 mg daily. The hallucinations now resembled more classic tinnitus, sounding like bells or sirens. This tinnitus varied in intensity according to the patient’s stress level or the environmental noise. Following a good evolution of the patient’s depression, the amitriptyline and lormetazepam were progressively reduced and stopped 8 months after the introduction of amitriptyline. The tinnitus regressed but remained as a slight whistling.

The patient’s medical history was simple, with no substance abuse. During the episode, and before the introduction of
amitriptyline, the psychiatric evaluation indicated moderate anxiodepressive symptoms, with a score of 10 on the Beck Depression Inventory and scores of 63 and 50, respectively, on the anxiety state and trait subscales of Spielberger’s State-Trait Anxiety Inventory. There were no personality or psychotic disorders. The neurologic examination was normal. An EEG done before amitriptyline treatment was normal, without evidence of epilepsy. The patient had never complained about hearing loss, and there was no recent audiometric testing.

Tinnitus and auditory hallucinations have been associated with benzodiazepine discontinuation (7,8). Musical hallucinations are only rarely related to benzodiazepine consumption. They were reported in a 57-year-old man in good physical condition after he stopped taking triazolam for 8 nights (9). Musical hallucinations were also reported in a 65-year-old woman whose tinnitus changed to musical hallucinations after the introduction of lorazepam and temazepam and then evolved into a rumbling noise when benzodiazepines were stopped (10).

In the absence of neurologic, otologic, or psychic disorders, the fact that the hallucinations began with the introduction of lorazepam and changed when the dosage was reduced with the introduction of amitriptyline suggests a causal relation to lorazepam. The pathophysiological mechanism remains obscure. A convulsive mechanism seems unlikely. Lorazepam’s 10-hour elimination half-life makes any withdrawal phenomenon between 2 drug administrations unlikely. An interesting element is the qualitative change in the hallucination with the introduction of amitriptyline, because tinnitus is reported by about 1% of patients receiving tricyclic antidepressants (11). The continued symptoms after the drugs were stopped could suggest that some undetected predisposing oto-neurologic factors facilitated the appearance of the hallucination.

References


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Bupropion-Methylphenidate Combination and Grand Mal Seizures

Dear Editor:

I report a case of grand mal seizures emerging in an adolescent boy being treated with combined bupropion and methylphenidate for attention-deficit hyperactivity disorder (ADHD) symptoms.

This 14-year old, 50-kg, white youth was brought to the emergency room following an episode of tonic-clonic movements, perioral cyanosis, and loss of consciousness. The episode lasted for more than a minute, and subsequently, the patient was somnolent and confused. He had no history of seizures or any other medical condition predisposing him to seizures.

The patient had been in treatment with methylphenidate 60 mg daily when bupropion was introduced to target dysphoric symptoms. The initial bupropion dosage was 200 mg daily, increased to 300 mg daily. The patient experienced the seizure 4 weeks after the dosage increase. Bupropion was discontinued, and the patient has been maintained on methylphenidate and remained seizure-free for 12 months.

Bupropion has been associated with seizures in adult patients treated for depression (1) and smoking cessation (2). Information about the risk of seizures in children treated with bupropion is limited. In a multisite, double-blind trial of bupropion in 72 school-age children, EEGs switched from normal to abnormal in 6 subjects (8.3%); none of the subjects experienced seizures (3). More recently, a case of seizures in a 10-year-old treated with combined bupropion and guanfacine was reported (4). Methylphenidate’s reputation of lowering the seizure threshold originates from animal studies in which massive amounts (300 mg/kg) were used to determine the lethal dose. Available evidence suggests that stimulant treatment for ADHD symptoms in children with epilepsy maintained on effective anticonvulsant treatment does not produce increased seizure frequency, EEG changes, or difficulty regulating blood levels of anticonvulsants (5,6).

It is quite likely that, in the case presented, seizure onset is linked primarily to bupropion. However, it is also possible that the risk of seizures was amplified by the combination of bupropion with methylphenidate. Clinicians should exercise caution when treatment with a medication known to lower seizure threshold is augmented with other psychotropics. When prescribing bupropion alone, or combined with other drugs in children and adolescents, patients and parents should be advised of the potential seizure risk.
The Association of Depressed Affect and Stroke in Institutionalized Canadians

Dear Editor:

Depression following stroke is common, with a reported prevalence ranging between 25% and 46% (1). Depression may adversely affect functioning, recovery, and survival and may increase the risk of stroke morbidity and mortality. The Canadian National Population Health Survey (NPHS) has 2 components: a household component and an institutional component. A previous analysis of stroke data from the NPHS was based exclusively on the household component, and the number of subjects reporting stroke and depression was too low to support a statistical analysis (2). Statistics Canada has now released the data for the institutional component and it is now possible to evaluate the association between stroke and depression in a national sample of institutionalized adults.

The target population for the 1996–1997 NPHS institution survey included all long-term residents of health institutions in Canada, excluding the territories, Indian reserves, and Canadian Forces bases (3). Data collection was carried out by personal interview, where possible, or by proxy interview, when required because of illness or incapacity (59.1% of interviews were completed by proxy). The sample consisted of 2118 individuals representing a target population estimated at 222 967. The institutional response rate for this survey was 100% (n = 213), and the individual response rate was 88.9%. We used a single item to evaluate depression: “How would you describe yourself as being usually?” Subjects who responded “unhappy, with little interest in life” and “so unhappy that life is not worthwhile” were regarded as suffering from depression. The survey interview also included a question asking, “Do you suffer from the effects of a stroke, such as paralysis or speech problems diagnosed by a health professional?” Finally, an item asked whether the subject had been diagnosed with a mental disorder by a health professional. Statistical analysis employed procedures recommended by Statistics Canada to account for the survey sampling procedures.

Very substantial proportions in this population reported depression and stroke: 16.1% and 20.1%, respectively. Depression was approximately twice as common in those with stroke (22.7%; 95%CI, 21.5% to 23.9%) as in those without stroke (12.8%; 95%CI, 10.9% to 14.6%). The association was equally evident in men and women and persisted after stratification for age (that is, over or under age 70 years).

The major weakness of the NPHS institution depression data is the measurement of mood status, which is based on a single self-report item. To address this, we identified the subset of those subjects reporting depression who also reported having had a mental disorder diagnosed by a health professional; 5.4% of persons with stroke fell into this category (95%CI, 3.5% to 7.3%), compared with 2.9% of those not reporting the effects of a stroke (95%CI, 2.1% to 3.7%). The rates are much different, but the approximate doubling of prevalence is still evident. This analysis confirms, as expected, that stroke is strongly associated with depression in residents of health care institutions in Canada.

References

displaying increased paranoia, loosened associations, and disorganized thinking and behaviour. On return to hospital, he had a temperature of 38.4°C, a fluctuating blood pressure as high as 160/110, and a rapid, regular pulse rate of up to 140 beats per minute. He maintained postures, his muscle tone was only minimally increased, and his creatine phosphokinase (CPK) was elevated to 1613 IU/litre. His attention was impaired, and his sparse verbal output was grossly disordered, exhibiting grandiose and paranoid themes. He was extensively investigated for a fever of unknown origin. Aside from an elevated white cell count of 15.9 x 10^9/litre and CPK levels that peaked at 3485 IU/litre, the remaining results were all within the normal limits. He was treated for NMS with rehydration and dantrolene until his fever, pulse, blood pressure, and CPK settled 3 weeks later. His CPK fell to below 1000 IU/litre within 3 days of discontinuing quetiapine and took another 2 weeks to normalize.

Since he remained mute, disorganized, incontinent of urine, and severely psychotic, he was transferred back to psychiatry. When treated with a course of unilateral electroconvulsive therapy, he improved after the first treatment, becoming more communicative and organized. He recovered after 7 treatments and was restarted on olanzapine 10 mg daily. He was discharged for outpatient follow-up and 1 year later remains well and stable in the community.

NMS has been reported as a rare complication of treatment with clozapine, risperidone (1), and most recently, olanzapine (2). Quetiapine is a novel dibenzothiazepine clozapine–like neuroleptic with fewer D2-blocking properties than clozapine (3). It has a correspondingly low propensity to induce extrapyramidal side effects (EPS) (4). There are 3 reports of possible NMS associated with quetiapine, 1 case of which occurred with concomitant use of loxapine (5), 1 with concomitant use of sulpiride (6), and 1 in which quetiapine was the sole antipsychotic (7). The NMS picture that occurs with atypical antipsychotic agents may be milder than that which occurs with typical agents (8) and corresponds to 2 of the reported cases involving quetiapine (6,7). This patient had mild physical manifestations and more prominent mental status changes. Despite quetiapine’s low propensity to cause EPS, NMS needs to be considered in the differential of patients on quetiapine who present with NMS-like features.

References

Kevin Solomons, MD, FRCPACan J Psychiatry, Vol 47, No 8, October 2002

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