LETTERS TO THE EDITOR

Moving From the Clinic to the Community: The Alone Mothers Together Program

Dear Editor:

Health promotion among lone-mother families should be a priority because they constitute a large segment of the population, experience extraordinarily high poverty rates, and are at elevated risk of depression and other mental health problems. Moreover, their health care use is suboptimal. These considerations stimulated our move from clinic-based support and education groups for lone mothers to community-based groups. The pilot study of community-based support and education groups for poor lone mothers of preschoolers focused on identifying and enlisting lone mothers, retaining them in the groups, completing evaluations, and designing acceptable evaluation tools.

In the planning period, we identified the need to collaborate with key community agencies, to get broad community exposure, and to provide a high-quality, structured children's program. More intensive advertising (for example, flyers and information sessions) was done in areas where groups were organized. Interested mothers registered by telephone or in person at information sessions. Enlistment was aided by providing an Ontario Works credit for participation, as well as snacks, transportation, and a clothing exchange. To sustain group involvement, mothers received telephone reminders and transportation assistance.

To facilitate evaluation, we provided flexible times, sites, and compensation for questionnaire completion. The evaluations indicated that measures were acceptable in terms of length and language. Self-report (1–5) and observer-rated (6) measures assessed maternal well-being and parenting. Evaluations were carried out both before and after the group sessions. During the pilot study, we organized 2 group interventions, each 8 weeks long. Community-based support and education groups run within subsidized housing projects met weekly for 1½ hours in sessions led by 2 experienced group leaders. Recurrent discussion themes included issues common to lone mothers—the experiences of poverty and social isolation—and child-rearing issues (for example, concerns about child development and behaviour management). A parallel children's program focusing on social, language, and motor skills was provided to the preschoolers. We developed a manual for each group.

Advertising efforts attracted 20 lone mothers who expressed interest, of whom 10 agreed to participate in the intervention. Eight of the 10 completed more than one-half the group sessions, and 6 of the 10 completed the postgroup evaluation.

We successfully achieved our pilot goals (that is, we identified and enlisted lone mothers into the groups, we maintained a satisfactory participation level, and we developed acceptable evaluation tools). This provided the basis for proceeding to a randomized controlled trial evaluating the effectiveness of these groups in the community. Despite strong expressed community support, recruiting mothers was an intensive process that attracted only small numbers. Readiness to change and to engage in treatment activities appears to be much lower in the community, compared with clinic settings, where mothers have been mobilized to ask for help with specific problems. Accordingly, those developing community-based programs must pay close attention to the tradeoffs between the intervention program's acceptability and attractiveness and the burden of participation defined by the intervention's intensity, duration, and structure.

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Citalopram Overdose, Serotonin Toxicity, or Neuroleptic Malignant Syndrome?

Dear Editor

We would like to comment on the letter "Neuroleptic Malignant Syndrome Due to Citalopram Overdose" (1): we question the authors' assertion that they present the first case of neuroleptic malignant syndrome (NMS) due to citalopram use. The reported case contains a few features that are possibly consistent with NMS, but it occurred after an overdose of an agent that is much more likely to cause serotonin toxicity. Serotonin toxicity is far more likely to occur with selective serotonin reuptake inhibitors (SSRIs) than is NMS, and we believe that this is what has been described.

The diagnostic criteria for NMS usually include 4 principal features: autonomic lability, hyperthermia (pyrexia) without other cause, extrapyramidal syndrome (commonly cog-wheel or lead pipe rigidity), and encephalopathy (2). In the patient described, there was no evidence provided of autonomic lability—only the initial heart rate and blood pressure were reported. The patient had a low-grade temperature on admission, but no other temperature measurements were reported. Only mild rigidity (of unclear type) of the limbs and abdominal musculature was described. Finally, the encephalopathy of NMS cannot be determined in a comatose patient (coma of itself is very unusual in cases of NMS). Thus, the patient had none of the major features of NMS at the time of the diagnosis. A raised creatine kinase (CK) is not diagnostic of NMS (2) and, in this patient, could have resulted from numerous other causes that were not excluded.

The patient was described as regaining consciousness 6 hours after bromocriptine administration. No details are provided, however, concerning sedation given or paralysis used while the patient was ventilated. Regaining consciousness was more likely to be part of the clinical course or the result of any sedation wearing off. Administering bromocriptine in cases of NMS will lead to resolution of muscle rigidity, autonomic lability, and hyperthermia; it will not particularly increase the level of consciousness alone.

We believe that the condition described in this case is more likely to have been serotonin toxicity, which is a recognised complication of SSRI overdose (3). Usually referred to as the serotonin syndrome, the condition is better described as a spectrum of toxicity (4). Although Sternbach's criteria are the most commonly cited for diagnosis (5), serotonin toxicity is more clearly characterized by a triad of clinical features: autonomic features, neuromuscular changes, and altered mental status (3).

Although there are similarities to NMS, serotonin toxicity is a distinct condition (6,7). NMS is an idiosyncratic reaction to therapeutic dosages of neuroleptic agents, while serotonin syndrome is a toxic reaction due to overstimulation of 5-HT_{2a} receptors in the central nervous system (6,8). Distinguishing features include bradykinesia and lead pipe rigidity in NMS, compared with hyperkinesia and clonus in serotonin toxicity, and autonomic instability in NMS, compared with presence of a serotonergic agent in serotonin toxicity (6,7). In the case described, there is evidence of serotonin toxicity; however, there are insufficient clinical details to confirm the diagnosis.

Over the last 2 years, there have been 46 citalopram overdose presentations to our unit. There was moderate-to-severe serotonin toxicity in 7 cases, and in 4 of these, citalopram was the only drug ingested. All 7 met Sternbach's criteria (5). The dosage ingested by the 4 patients using citalopram alone ranged from 280 mg to 3000 mg daily. There were no cases of NMS. This suggests that serotonin toxicity is not uncommon with citalopram overdose. There is 1 report in the literature of serotonin syndrome caused by an infusion of 20 mg of citalopram (9).

Because the described clinical features did not satisfy the diagnostic features of NMS (2), other differential diagnoses should have been considered by the authors—in particular serotonin toxicity. It is our contention that the authors need to make an unusually strong argument for the case of citalopram-induced NMS: the patient they describe had a citalopram overdose with very few features of NMS. We believe this is not a case of NMS due to citalopram overdose; it is more likely to be the expected serotonin toxicity.

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Reply: Citalopram Overdose, Serotonin Toxicity, or Neuroleptic Malignant Syndrome?

Dear Editor:

We are grateful to your correspondent for opening the debate on our letter.

Neuroleptic malignant syndrome (NMS)—like the encephalopathy that develops in association with the use of antidepressants—indicates that both it and serotonin syndrome are spectrum disorders induced by drugs with both antidopaminergic and serotonergic effects (1). Medications that affect brain dopamine and serotonin levels are occasionally associated with neurotoxic behavioural and autonomic syndromes, variously described as NMS and toxic serotonin syndromes (TSS). Based on the drugs' presumed brain mechanisms of action, different corrective interventions have been recommended. Moreover, Fink has claimed that NMS and TSS are not specific syndromes but, rather, examples of a nonspecific generalized neurotoxic syndrome and that they are

subtypes of catatonia (2). For these reasons, NMS and TSS may be confused—or they may be presentations of different aspects of the same etiologic cause (dopamine–serotonin imbalance).

Although NMS is described as having 4 classic signs, no agreed-upon criteria exist for the diagnosis of the syndrome in terms of severity or combination of these signs, and milder or incomplete varieties have been detected and included with the full-blown cases (3). In our patient, there was no autonomic lability, but there was coma, subfebrile fever, and rigidity on admission. In NMS, mental status changes, coma, and catatonia are common (4). Although creatine kinase (CK) is not a specific indicator of NMS, it may be important for the early detection of the syndrome (3). Muscle isoenzyme of CK may be raised by intramuscular injections, hyperactivity, and catatonia (5); it may also be raised in medically ill patients taking neuroleptics (6). In our case, there were no such conditions.

No sedation or paralysis agents were used while the patient was ventilated. Therefore, consciousness was not regained as the result of any sedation wearing off. Otherwise, regaining of consciousness was not the part of the clinical course, it was a secondary event to bromocriptine administration. We observed no change during a 7-day period of supportive treatment, but the clinical course improved completely within 24 hours of bromocriptine administration. In one review, it has been stated that, when compared with supportive measures alone, bromocriptine in particular shortened the time to the resolution of NMS symptoms (7). Since there was neither hyperkinesia nor clonus in our case, we did not suspect serotonin toxicity.

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Lycanthropy: 2 Case Reports

Dear Editor:

According to Keck and others (3), lycanthropy can be diagnosed when a patient reports in a lucid moment, or retrospectively, feeling, or having felt, sensations of being an animal. Similarly, it can be diagnosed when a patient displays animal-like behaviour (for example, howling or crawling). It does not form a separate syndrome but is a symptom of different psychiatric diseases (2). We report 2 cases of patients with schizophrenia who felt they had been transformed into a frog and a bee, respectively.

Case Report 1

A 34-year old woman suffering from schizophrenia came to the emergency department. At first, she was mutistic; later she seemed agitated and tense. Suddenly, she started moving like a frog, jumping around, making frog-like noises, and showing her tongue as though intending to catch a fly. We found out that she had taken part in a workshop about fairy tales prior to becoming symptomatic. An organic cause was excluded, and no drug intake was found.

Case report 2

A 24-year-old woman suffering from schizophrenia reported a strange feeling that could not be properly described, together with the feeling that she was becoming a bee and getting smaller and smaller. She also felt a burning sensation in her thighs. She attributed her metamorphosis to her being stung by bees as a child and the "splashes of bee" that had touched her. In this case, it is interesting to note that she described herself as caring for her mother like a "working bee" and that she could not really develop autonomy. No organic causes could be found, and drug screening tested negative.

Discussion

To date, the metamorphosis into a frog or a bee has not been described in the medical literature. Both patients suffered from schizophrenia, and the lycanthropy was accompanied by other psychotic symptoms. Psychodynamically, lycanthropy could be an attempt to delegate affects to the animal. Lycanthropy in our cultural context seems bizarre and strange, appears suddenly, and does not seem to be understood rationally. In his discussion of countertransference phenomena induced by lycanthropy, Knoll points out that its strange and often threatening aspect may lead therapists to neglect it (3). This might explain why the phenomenon is rarely described in psychiatric literature. Focusing on the patient's subconscious choice of animal species may hint at the patient's subconscious conflicts and might be helpful in psychotherapy of the mental disease.

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Re: Weight Change With Antipsychotic Use

Dear Editor:

Weight increase with the use of antipsychotics, especially novel antipsychotics, has been of significant concern to patients suffering from psychotic disorders. There are several psychosocial issues associated with weight gain (for example, issues of self-esteem, discrimination, and medical morbidity). Studies and clinical practice involving antipsychotic use consistently indicate weight gain as a potentially serious side effect.

It has been stated that weight gain and loss may not be dosage-related. In my clinical observation on 2 different occasions, however, dosage appeared to be related to weight loss after initial weight gain. I present here the cases of 2 patients who lost weight, following initial gain.

Case Report 1

A 25-year-old man suffering from schizophrenia was prescribed olanzapine 20 mg daily (monotherapy). His weight was recorded at 100 kg. He was clinically stable. The dosage was reduced to 17.5 mg daily, and then to 15 mg daily, with no rebound increase in psychosis. His recorded weight showed a reduction of 7 kg, accompanied by more self-confidence in the patient. There was no other change in his medication regimen, diet, medical condition, lab values, or daily routine.

Case Report 2

A 37-year-old lady suffering from schizoaffective disorder with manic and psychotic symptoms had a trial of several mood stabilizers, typical antipsychotics, long-acting antipsychotics, and clozapine, with multiple subsyndromal to full-blown symptoms and frequent hospitalisations. She was treated with 75 mg of topiramate and was started on olanzapine with dose titration and final increase to 20 mg daily. She experienced a weight gain of 11 kg in 3 months. This combination of mood stabilizers and olanzapine led to significant improvement in her mental state. Initially, it was a daily struggle keeping her on olanzapine, because she was extremely weight-conscious. With improvement in her clinical condition, olanzapine dosage was decreased to 17.5 mg daily, and then to 15 mg daily, with weight reduction of 3 kg. She was much more accepting of olanzapine and was discharged.

Based on these observations, it is not possible to determine the mechanism of action leading to weight loss. Olanzapine dosage reduction—wherever possible at the earliest opportunity—may, however, be a useful strategy to reduce weight following initial gain.

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Misleading Drug Advertising

Dear Editor:

It must be evident to all psychiatrists of my vintage (over age 65 years) that pharmaceutical company advertising has changed remarkably in the last 40 years. In the 1960s, battling the hegemony of psychodynamically oriented psychiatry, the drug companies timidly promoted their products (sometimes, even the neuroleptics) as adjuncts to psychotherapy (1).

Now, we seem to be in an era wherein the aim of drug advertising is to devalue a competitor's product in the hope of casting a favourable light on your own. For example, we have the altruistic folks at the Lilly Company informing us in a 2-page advertisement (2) of the dangers of prolonging the QTc interval, possibly in anticipation of ziprasidone's introduction by a competitor. We also have the thoughtful people at Janssen telling us about the high incidence and dangers of type 2 diabetes, an increased risk for those treated with olanzapine (3).

There is nothing wrong with receiving factual information. Every clinician knows that the use of any drug for a particular patient requires the exercise of sound judgement and a careful balancing of advantages against disadvantages. When drug companies try to deceive us, however, they lose credibility; unfortunately, their spokespersons may also lose credibility. The following 2 examples are illustrative.

The March 2001 issue of *Physician Perspective*, a newsletter sent through the mail by Eli Lilly Canada Inc, contained the "viewpoint" of Canadian physician, Joel Raskin, entitled "The New Atypicals and Abnormalities in Glucose Metabolism: Real or Exaggerated Risk?" Dr Raskin, who just happens to be the vice-president of clinical research for Lilly, states: "Risperidone and ziprasidone were designed to behave like haloperidol, but without giving rise to extrapyramidal symptoms." Anyone aware of the pattern of receptor blockade of these 2 drugs, and that of haloperidol (and others, for example see [4] and [5]), will find this statement grossly misleading. A recent article by Kapur and Seeman (6) is also relevant.

A second example: A friend of mine recently picked up a pamphlet in her family doctor's office. *Anxiety—It's Not Just In Your Head* was authored by Michael Evans, MD, and Richard Swinson, MD. Its publication was "supported by an educational grant from Wyeth-Ayerst Canada Inc," the makers of Effexor (venlafaxine). The pamphlet is structured as a series of questions the prospective patient may have. Question 13 reads as follows: "Are the medications addictive? Antidepressants are not addictive in the same way that cigarettes or benzodiazepines are." To suggest that the addictive potential of benzodiazepines is comparable with that of

nicotine (cigarettes) is deceitful and serves only to frighten unnecessarily those patients who have been helped by benzodiazepines.

Can drug companies not do better than this? Psychiatrists are not stupid.

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