Letters to the Editor

A Novel Form of Treatment Resistance in Anorexia Nervosa

Dear Editor: Treatment refusal, which often involves resistance to feeding, is common in patients with anorexia nervosa (AN) (1–5). We report the first case of a patient with AN who tied a knot at the gastric end of a feeding tube to avoid being fed.

Case Report
A single woman, aged 19 years, was admitted as an emergency to the medical ward of our hospital for feeding and medical management of severe malnutrition, dehydration, and acute renal failure due to AN. Her admission height and weight were 172 cm and 37.7 kg (body mass index 12.7 kg/m²). One year before admission, her weight had been 63.3 kg (body mass index 21.4 kg/m²). Over the year, she had increased her exercise, decreased her food intake, and increased her use of laxatives. We estimated that her average daily food intake for the last 3 months was 300 to 500 kcal. During the week before admission, she experienced weakness, persistent light-headedness, and difficulty completing her usual activities of daily living.

After admission, her electrolyte and urea levels returned to normal, but she did not gain weight. She began nasogastric tube feeding on the fourth day of hospitalization, with her daily caloric intake being increased to 2480 kcal. Her anxiety increased coincident with increased feeding, and she complained of losing control. The nasogastric tube was flushed regularly with sterile water, but during one of the scheduled flushes, the nurse was not able to push water through the tube. Numerous attempts to reestablish the patency of the tube by exerting increased pressure and with infusion of pancreatic enzymes failed. When the tube was removed for inspection, a knot was present at the tube’s distal end. The knot was partially light-headedness, and difficulty completing her usual activities of daily living.

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The patient would have had to remove the tube, tie the knot, and reinsert it herself to make a knot at its gastric end. Normally, blockage at the distal end of a feeding tube is assumed to be caused by solidification of the formula. If attempts to clear an apparently blocked nasogastric tube fail, the tube should be removed and closely inspected for the presence of such a knot.

References

Andrei V Krassioukov, MD
C Laird Birmingham, MHSc, MD, FRCPC, FACP, ABIM
Vancouver, British Columbia

Capgras Syndrome in the Modern Era: Self Misidentification on an ID Picture

Dear Editor: While living in a supervised apartment, Ms KL, aged 31 years, was hospitalized for a psychotic relapse in the course of undifferentiated schizophrenia. At admission, she presented with intense delusional (mostly persecutory) ideation, auditory hallucinations, severe anxiety, and mild depression. Her behaviour was grossly disorganized, and she required close supervision to tend to daily life activities. Her drug treatment was changed from risperidone 4 mg daily to olanzapine 30 mg daily, which elicited a partial symptom remission. Further improvement occurred with the addition of venlafaxin 225 mg daily, but she remained symptomatic and severely impaired, which led to the discussion and proposal of clozapine treatment, an option that, as of this report, she has refused.

During the course of the olanzapine and venlafaxin treatment, the patient presented with a new clinical relapse, including notably increased persecutory ideation and different components of Capgras syndrome. First, she discarded her health insurance (RAMQ) plastic card because she thought that its photograph was not her own and, more specifically, because she did not recognize her nose. Of note, the patient had held this card for years without particular concern. When rechallenged with the photograph, she still contended that it had been replaced or tampered with and did not recognize her face. Second, she also discarded various personal objects (such as her satchel, clothes, toilet items, and magazines) because she was convinced that they were not hers. She retrospectively admitted that she had already done so in the past; however, this behaviour was not active in the hospital before her relapse. Third, she became suspicious of her roommate, although with fluctuating conviction, because she thought that the latter had been replaced by someone charged with killing her.

While the second and third symptoms are fairly classic, this case adds the plastic ID card, a highly meaningful feature of contemporary life, to the long list of objects (in a broad sense) misidentified in Capgras syndrome: it stands at the limit of object (in a narrow sense), face, and self-recognition. Moreover, the quality of most ID photographs is poor (which was admitted particularly pronounced in the present case), and the patient was therefore presented with a degraded picture, the recognition of which is notably difficult for patients with schizophrenia.

Because we had the opportunity to assess the occurrence of Capgras syndrome in an emerging state, we would also like to speculate on its clinical course in patients with schizophrenia. Visual, and especially facial, recognition difficulties are enduring features of schizophrenia, and some authors have suggested that these may be trait markers of the disease, as well as endophenotypes with potential use in genetic research (1,2). Conversely, Conklin and others have suggested that impaired recognition memory for faces is associated with increased positive symptoms in patients and increased schizotypal traits in their relatives (3). In the case discussed here, it can be argued that
there was some stable level of visual recognition impairment; a fully developed Capgras syndrome, however, only appeared in association with a psychotic relapse. Within the framework of the “salience theory” of psychosis (4,5), this suggests that increased dopamine mesolimbic neurotransmission associated with psychotic relapse adds salience, novelty, and in this case, aversive features, to a familiar, albeit misidentified, visual stimulus that would otherwise have remained neutral in a remitted patient. Once present, however, Capgras syndrome shares the stability and refractoriness of the underlying cognitive deficit.

References


Sylvain Grignon, MD, PhD
Mikael Trottier, Medical Student
Sherbrooke, Quebec

Effectiveness of Risperidone in Delirium

Dear Editor: Delirium is a common and complex neuropsychiatric syndrome seen frequently in medical settings. If untreated, it is associated with high mortality (1). Apart from a multifaceted interdisciplinary approach involving environmental strategies, antipsychotic medication remains the cornerstone of treatment (1). Among these drugs, the typical antipsychotic haloperidol has been the drug of choice, mainly owing to its lack of anticholinergic (1) and minimal hemo dynamic and respiratory side effects (2). Over the last decade, however, evidence has emerged for the use of second generation antipsychotics like risperidone—but only in the form of case reports and case series (3,4).

We report our experience in treating 7 cases of delirium with risperidone.

To identify the target group, we screened case notes of all patients with a diagnosis of delirium seen over a 4-year period. Seven patients who had delirium when assessed were treated with risperidone.

The demographic profile was as follows: the patients’ mean age was 32 years (range 24 to 67 years); 60% were men, and 40% were women; they had on average 6 years of education; and 70% had low socioeconomic status. There was only 1 elderly woman (aged 67 years) with past cardiac problems and current hypoxic delirium. The rest of the sample had no past cardiac problems and presented with hyperactive delirium. The mean duration of delirium before initiation of treatment was 5.29 days (range 1 to 15 days, with the maximum for the elderly woman). All subjects were treated with environmental measures, correction of the underlying cause, and risperidone. The dosages of risperidone were as follows: average starting and maximum dosage was 1.14 mg daily (range 0.5 to 2.0 mg daily); the average dosage through the period of treatment was 1.07 mg daily (range 0.5 to 1.5 mg daily). All patients were closely monitored (at least once every 48 hours.) No subject developed observable extrapyramidal or any other serious side effects. All patients, except for the elderly woman, were either significantly improved or recovered at the last follow-up. This small, open-label, retrospective case series demonstrates that risperidone in low dosages is effective and safe for treating delirium.

Risperidone possesses certain advantages over haloperidol, specifically, fewer extrapyramidal and cardiac side effects, including a reduced propensity to cause QT prolongation (5). This profile, along with a low-dosage regime, gives it a unique advantage over haloperidol. However, the current lack of an available intravenous parenteral formulation, as well as the lack of a large database on risperidone use in treating delirium, may preclude its use as a standard or textbook treatment modality. Controlled, double-blind, prospective studies are warranted to establish the efficacy of risperidone as a useful alternative to haloperidol in treating delirium.

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References


Nitin Gupta, MD,
Burton upon Trent, UK
Parveen Sharma, MD,
Surendra K Matteo, MD,
Chandigarh, India

Family-Oriented Rehabilitation for Unexplained Chronic Pain

Dear Editor: We present a case report to illustrate our experience with a family-oriented rehabilitation program for children and adolescents suffering from unexplained chronic pain.

Case Report

A girl, aged 12 years, had a 6-month history of unexplained abdominal pain described as a “hurt feeling inside,” fatigue, decreased concentration, sleep difficulty, and school avoidance. She was described as an excellent student and a perfectionist in all her activities. Consultations from a pediatrician, gastroenterologist, neurologist, and allergist ruled out significant organic pathology to explain her symptoms. Her parents were angry that a pediatric gastroenterologist, neurologist, and allergist ruled out significant organic pathology to explain her symptoms. Her parents were angry that a medical cause was not found and denied the existence of psychosocial stressors. The child’s aunt had recently died from a tumour that presented with unexplained pain symptoms for months prior to diagnosis.

Psychometric testing revealed an anxious coping style and perfectionist traits. During the assessment, the family expressed their feelings of anger about their aunt’s death and began to confront their unresolved grief. The patient began taking fluvoxamine 100 mg every night. As a goal of rehabilitation, we emphasized enhanced functioning, specifically, improved sleep, increased activities,
and school attendance. We encouraged the parents to support these goals. After 3 weeks, the patient’s sleep improved, and her fatigue and pain decreased. She went to overnight camp for 2 months, returned home, and attended school. Monthly family meetings continued for 6 months. Fluvoxamine was discontinued, and the patient continues to be well at 9 months.

**Discussion**

Pediatric unexplained chronic pain (UCP) presents a diagnostic and management challenge. Typically, families believe that UCP has solely an organic cause and frequently resist suggestions that psychosocial factors may be responsible for its development or perpetuation (1,2,6). Consequently, such patients develop a pattern of help-seeking behaviour involving multiple diagnostic investigations, repeated emergency department visits, hospital admissions, and prolonged hospital stays (3). Family physicians, pediatricians, and specialists in gastroenterology, rheumatology, endocrinology, and neurology are consulted frequently.

To address this problem, we have established a multidisciplinary, family-oriented, and rehabilitation-focused program to treat patients with UCP. We use a battery of psychosocial self-report scales to evaluate perfectionism, anxiety, and depressive symptoms (4,5,7). Treatment goals focus on enhanced patient functioning (with emphasis on sleep, school attendance, and social interaction), rather than on the symptoms. The family is taught coping strategies for enhanced functioning. Existing similar programs are also oriented to coping and function rather than the pain symptoms (1). A selective serotonin reuptake inhibitor is added to address anxious coping.

In a pilot study evaluating this program, we administered self-report scales to 15 patients (5 boys and 10 girls; mean age 12.4 years, SD 3.2) with a minimum of 3 months of UCP. The referring physician excluded significant organic pathology. Overall, we found that anxious coping styles and difficulty with separation were common among the referred population. Levels of perfectionism fell between those in a healthy control group (low) and those in a group with anorexia (high). Elevations occurred in the domains of feeling unsatisfied after completing projects, high personal standards, and the need for order. These results were psychoeducational in that they helped parents and patients focus on the rehabilitation goals and enhanced their confidence in a program that targets function, rather than pain.

**References**


Gillian Kirsh, MA
Rose Geist, BSc, MD, FRCPC
**Toronto, Ontario**

**Hypokalemia from Risperidone and Quetiapine Overdose**

**Dear Editor:** Hypokalemia has been listed as one of the known toxic effects of both quetiapine and risperidone. It is a risk factor for potentially life-threatening arrhythmias like torsades de pointes, caused by a prolonged QT interval—another side-effect of antipsychotic drugs, including quetiapine (1). There is a solitary report of mild hypokalemia following a quetiapine overdose of 9600 mg (2). Here, we report a case of remarkable hypokalemia following a much smaller dose of quetiapine and risperidone.

**Case Report**

Mr B, a white man aged 44 years, had a diagnosis of bipolar disorder not otherwise specified. He was taking risperidone 3 mg daily, lithium carbonate 900 mg daily, and quetiapine 50 mg at bedtime. At about 20:30 hours on the night of presentation, he attempted suicide by taking approximately 750 mg of quetiapine and between 90 and 120 mg of risperidone.

He denied taking lithium in his attempt. This was substantiated by a subsequent test that revealed a lithium level of 0.4 mmol/L.

He presented to the emergency room about 2 hours after the ingestion and was stabilized medically. Initial labs drawn at the time of emergency department presentation showed potassium levels of 2.9 mmol/L and magnesium levels of 1.7 mg/dL (with lower normal levels being 1.8 mg/dL). His potassium level was initially corrected with intravenous cocktails in the emergency department and intensive care unit and was followed over the next few days. It subsequently plateaued and remained stable and in the normal range. Mr B recovered fully and was discharged home after a brief hospital stay.

**Discussion**

Both risperidone and quetiapine can cause hypokalemia. However, at about 350 mg or more, the risperidone dose reported to cause hypokalemia is much higher than that taken by Mr B. The previously reported case of hypokalemia following quetiapine overdose found a potassium level of 3.3 mmol/L after ingestion of 9600 mg (2). Our patient ingested only about 750 mg of quetiapine and about 100 mg of risperidone. Could it be that the drugs acted synergistically to cause a more dramatic hypokalemia? Although exact mechanisms of action are not clear, it is possible that both drugs cause a more dramatic drop in serum potassium following acute ingestion, which would further increase the chances of torsades de pointes and sudden cardiac death secondary to concomitant QT interval prolongation.

This case demonstrates that we need to be vigilant after an apparently nonlethal overdose of 2 drugs having a relatively safe overdose profile, both for possible synergistic effects of the 2 drugs on serum electrolytes and for the possible ensuing complications.

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**References**

A Renewed Interest in Day Treatment

Dear Editor: Day treatment, a form of partial hospitalization, can be helpful for patients who do not require inpatient care but who may benefit from more intensive care than is possible for outpatients. It differs from other forms of partial hospitalization (that is, day hospital and day care) in that it emphasizes both treatment and rehabilitation. Treatment is concerned with alleviating symptoms and recovery from illness. Rehabilitation focuses on the patient’s adapting to a disability and facilitates adaptive functioning in the community.

Historically, day treatment programs were offered to patients who were in some degree of remission from acute psychotic illness or to patients who suffered from long-term disorders (for example, personality disorders). Day treatment was viewed as superior to outpatient care because it provides more intensive treatment and rehabilitation. It experienced considerable use from the 1950s to the late 1980s. However, day treatment declined in the 1990s owing to inadequate funding arrangements and a move toward assertive community treatments.

Recently, this trend has reversed. Contributing to the renewed growth of day treatment is the recognition that, while many currently available treatments effectively reduce symptomatology, they often have minimal impact on functional impairments. This has contributed to high rates of relapse and recurrence. Multimodal treatments that focus on reducing illness and enhancing functional capacity are believed to offer an optimal intervention approach. Day treatment is seen as satisfying this need. It offers intensive and structured clinical services within a stable therapeutic milieu that typically incorporates group psychotherapy, biological psychiatry, milieu principles, and a systems orientation.

Many of the day treatment programs that have recently evolved differ from those used in the past. The newer day treatment programs are guided by the principles of cognitive-behavioural therapy; nearly all new programs incorporate some insight-oriented interventions. Finally, the application of day treatment has expanded beyond the patient populations it served in the past. It is now being used for mood disorders (1), obsessive–compulsive disorder (2), postnatal depression (3), eating disorders (4), and substance abuse disorders (5). This is not to say that the field has abandoned day treatment for patients with longer-term difficulties. Indeed, several authors have argued for longer-term day treatment for patients with personality disorders, because their response to shorter-term programs tends to be less than optimal. Considerable empirical evidence exists for the use of day treatment for personality disorders (6).

With renewed interest comes a renewed call for more empirical research on this form of treatment. There is considerable need for studies to identify the types of disorders best suited to day treatment. Demonstration of clinical effectiveness and cost-effectiveness relative to usual outpatient care is also urgently required. Hopefully, with greater empirical support of day treatment, clinicians and administrators will further recognize its unique advantages for treating many of the debilitating and recurrent illnesses encountered in most outpatient settings.

References


John S Ogrodniczuk, PhD
Vancouver, British Columbia
Paul I Steinberg, MD
Edmonton, Alberta

Quetiapine Therapy for Corticosteroid-Induced Mania

Dear Editor: Corticosteroids are routinely used for immunosuppression in patients who have received liver transplants (1). Mood symptoms and psychosis have long been documented as potential adverse effects of corticosteroid treatment (2). We report a case of corticosteroid-induced mania that followed a liver transplant and that resolved with quetiapine therapy.

A white man, aged 52 years, was admitted for a liver transplant. The donor was his biological brother. Approximately 5 years previously, he had been diagnosed with hepatitis B and hepatitis C. He had no psychiatric history; however, his daughter had been diagnosed with bipolar disorder. After the transplant, the patient was given methylprednisolone 50 mg intravenously every 6 hours (4 doses) and then 40 mg intravenously for an additional 4 doses. Cumulatively, he received more than 250 mg of steroids before the onset of behavioural symptoms.

On postoperative day 3, psychiatry was consulted because he was talking incessantly, preoccupied with hyperreligious themes, and making hypersexual comments. He claimed that he was a prophet and that he could speak different languages; his speech contained numerous neologisms. He was grandiose and claimed to possess spiritual powers that enabled him to “sense different pleasant odours.” He refused to divulge his name for “security reasons.” He had not slept for the last 2 days. He was not physically aggressive or agitated. The Young Mania Rating Scale (YMRS, 3) was used to assess symptom severity; his total score was 31, indicative of mania.

On a mental status examination, he was poorly groomed and disrobed repeatedly, but he was awake, alert, cooperative, and oriented as to place, person, and time. He displayed pressure of speech and rambling speech with incoherent narration, neologisms, and loose associations. His mood was significantly elevated and euphoric. His thought content revealed grandiose delusions, ideas of reference, and hyperreligious themes. His attention, concentration, insight, and judgment were poor.

The presumptive diagnosis was steroid-induced mania. A CT scan of the head on postoperative day 3 was unremarkable. He had a benign neurological examination; there was
no evidence of infection; and laboratory values were within normal limits. The patient was started on quetiapine 25 mg at bedtime and 12.5 mg as needed twice daily. Within 10 hours of quetiapine therapy, his mental status gradually improved. His YMRS score was less than 5 at discharge. His thought process and concentration improved significantly. He recognized that he had experienced changes in his behaviour and apologized to the staff. His sleep normalized, and he was discharged on the fifth day after his transplant. His tacrolimus level ranged from 21 to 20 by the time of discharge. His medications at discharge included acyclovir, prednisone 20 mg daily, and quetiapine 25 mg daily. The patient’s symptoms of mania did not recur, nor were there symptoms of depression during the subsequent 3 months. At the last follow-up visit, the results of his mental status examination were normal.

To our knowledge, this is the first case report of quetiapine use in treating steroid-induced mania in a transplant patient. This patient was given quetiapine for several reasons. It has antimanic and mood-stabilizing properties (4), as well as sedative properties. Its relatively short half-life and transient dopamine D₂ receptor binding are also to be considered in treating transplant and other patients with complex medical problems. Additionally, quetiapine may be administered frequently as needed for agitation and aggression. Interestingly, while this patient did not have any psychiatric history, he might have been vulnerable for developing mania, owing to his positive family history; possibly, he had an undiagnosed or subsyndromal bipolar disorder that was unmasked or exacerbated by steroids.

We conclude that quetiapine is a useful medication to consider for patients with steroid-induced mania. Its prophylactic use might be considered for patients at risk of developing a manic syndrome with steroids.

References


Zakaria Siddiqui, MD
Sriram Ramaswamy, MD
Frederick Petty, MD

Omaha, Nebraska