Letters to the editor

A CASE REPORT OF COTARD'S SYNDROME

DEAR EDITOR:

Cotard's syndrome is a relatively rare condition that was first described by Dr. Jules Cotard in 1882. Cotard's syndrome comprises any one of a series of delusions that range from a belief that one has lost organs, blood, or body parts to insisting that one has lost one's soul or is dead.[1]

Cases have been reported in patients with mood disorders, psychotic disorders, and medical conditions. Most cases of Cotard's are more responsive to electroconvulsive treatment (ECT) than to pharmacological treatment. We present the case of a recent immigrant with Cotard's syndrome, in the context of depression, to illustrate both how impairing the condition can be and how a course of effective, individualized therapy can improve outcome.

Case report. Ms. L, a 53-year-old Filipino woman, was admitted to the psychiatric unit when her family called 911 because the patient was complaining that she was dead, smelled like rotting flesh, and wanted to be taken to a morgue so that she could be with dead people. Upon interview in the hospital, the patient expressed fear that "paramedics" were trying to burn down the house where she was living with her cousin and her brother. She also admitted to hopelessness, low energy, decreased appetite, and somnolence.

Ms. L reported that she had been on antidepressants while in the Philippines (where she had resided for the last 18 years, having moved to the US only a month ago), but could not recall the name or dosage of the medication.

After organic causes were ruled out, treatment with quetiapine and bupropione SR was started. The patient was initially reluctant to take medication or eat. She subsequently developed an electrolyte imbalance (hypokalemia and hyponatremia), which necessitated intravenous electrolyte repletion. The patient was also isolative, spending much of the day in bed and neglecting her personal hygiene and grooming.

With her family's support, the decision was made to take the patient to court for treatment over objection. Subsequently, the patient's medication regimen was bupropion SR and olanzapine (intramuscular if she refused the oral form). A few days later, the patient had a questionable syncopal versus seizure episode, necessitating transfer to a medical unit.

After three days, she returned to the psychiatry floor where her medication regimen included olanzapine, escitalopram (because of the questionable seizure on bupropion), and lorazepam (for agitation).

Ms. L showed improvement in symptoms over one month on olanzapine 25mg daily, escitalopram 20mg daily, and lorazepam 2mg daily. At discharge she denied nihilistic or paranoid delusions and hallucinations and expressed hopefulness about her future and a desire to participate in psychiatric follow-up care.

Discussion. Previous reports of patients with Cotard's syndrome have indicated that ECT has tremendous advantages in resolution of patient's symptoms when pharmacotherapy has failed.[2,3] In contrast, Ms. L responded well to pharmacological medications at lower doses than previously needed for this degree of illness.

The family was supportive of the medical team's decision to take the patient to court for treatment over objection. Additionally, the family made daily visits, during which they attempted to encourage her to eat food that they had prepared. Other case studies have shown self-starvation to be associated with Cotard's syndrome.[4] This has legal implications since it fulfills the criteria for danger to self and sometimes necessitates involuntary commitment[4] and, in this case, treatment over objection.

We believe incorporating guidelines from a recent study on the mental health of Filipino Americans, which advocate utilizing family members in treatment of the patient and judicious use of medications due to the observed response of Asians at lower doses of medications,[5] contributed to our success.

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With regards,
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DISCLOSURES

Dr. Ruminjo is an AstraZeneca APA Minority Fellow; Dr. Mekinulov is a consultant to AstraZeneca and Eli Lilly.

NEUROENDOCRINE CARCINOID CANCER ASSOCIATED WITH PSYCHOSIS

DEAR EDITOR:

We report a case of an episode of psychosis in a patient with no known psychiatric history who was diagnosed with neuroendocrine carcinoid cancer. The case raises the possibility of a link between carcinoid cancer and psychotic symptoms. The case also discusses the treatment options of psychosis in a patient with carcinoid cancer.

Case report. A 62-year-old man with no known psychiatric history was admitted to an inpatient psychiatric unit for symptoms of paranoia and agitation that appeared one month earlier. He was diagnosed with neuroendocrine carcinoid cancer three months prior to admission after having complaints of persistent nausea. On admission, the cancer also spread to his liver and spine.

He presented for admission to the psychiatric inpatient unit for worsening psychotic symptoms, which included feelings that his family and neighbors were trying to hurt him and that people in the neighborhood were talking about him behind his back. The psychotic symptoms started two weeks before his admission. His symptoms also included anxiety with decreased sleep, psychomotor agitation, and irritability. He denied any hallucinations or perceptual disturbances. There was no prior psychiatric history of psychosis. The patient did not receive any psychiatric medications and was not on any psychotropic medications prior to admission. His admission labs were within normal limits. He was receiving weekly octreotide acetate injections that he had been receiving for two months.

On admission, the patient was started on quetiapine that was titrated to a dose of 100mg per day. The patient improved over the course of a week as he slept better, denied paranoid delusions or ideas of reference, and was less anxious. He was discharged home on quetiapine 100mg at bedtime.

Discussion. The overall incidence of carcinoid cancer in the United States is estimated to be 1 to 2 per 100,000 people a year.[1] The tumors are typically diagnosed in the fifth or sixth decade of life, and many patients are asymptomatic at presentation. In the patient with carcinoid tumor, tumor cells manufacture serotonin. Carcinoid syndrome is characterized by flushing, diarrhea, and abdominal cramping and occasionally by wheezing, heart-valve dysfunction, and pellagra. The incidence of the syndrome is higher with metastatic disease.[2]

Psychiatric symptoms have been reported in patients with metastatic carcinoid disease.[3] The reported frequency of depression in carcinoid patients varies widely from 50 percent to less than one percent among all patients in two different studies.[4,5] Carcinoid syndrome has been associated with psychosis in two case reports in the literature.[6,7]

There are multiple ways that carcinoid tumors can precipitate psychosis. Possible mechanisms include the hormonal effects of the tumor and of treatment with octreotide and the possible effects of systemic metabolic dysfunction. For example, elevated levels of the neurotransmitter serotonin (as occurs in carcinoid cancers) have been implicated in the pathophysiology of psychosis.[8] In addition, niacin synthesis is deficient in carcinoid syndrome because of metabolic diversion of its precursor, tryptophan, to form serotonin. In some untreated individuals this can even lead to pellagra, which has been associated with psychosis.[9] However, the precise mechanism is unknown.

This case is unique in the fact that an atypical antipsychotic was used to treat the psychotic symptoms effectively and without side effects. Atypical antipsychotics including quetiapine have serotonin receptor activity and have been linked to serotonin syndrome.[10] In this case report, the patient tolerated the medication and did not show any evidence of serotonin hyperactivity.

This case report adds to the existing literature by suggesting a possible link between the onset of new psychotic symptoms and a diagnosis with carcinoid cancer. The case highlights the need for awareness of the possibility psychotic symptoms in carcinoid cancer and that atypical antipsychotics can be effective and safe in the treatment of such symptoms.

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ABOUT CATATONIA IN ADOLESCENCE

DEAR EDITOR:

I read the excellent article titled "Catatonia in Adolescence: A Case Report" in the April, 2008, issue of your esteemed magazine. The article described an interesting case of an adolescent with catatonic features. The article mentions the principles of management of catatonia in Table 2 and treatment of catatonia in Table 3. Both the tables mention several important aspects of treating catatonia. The article correctly identifies benzodiazepines as the treatment of choice for catatonia. However, I would also add that zolpidem has been used successfully in the treatment of patients with catatonic features. Zolpidem has been documented in literature as another entity to alleviate catatonia.[1–3] This may be related to zolpidem producing effects on alpha -1 isoform of $GABA_A$ receptor. It has been postulated that zolpidem causes $GABA_A$ stimulation, leading to prefrontal cortex inhibition and activation of supplementary motor areas by thalamo-cortical circuits.[4] This may lead to improvement in catatonic patients. Also, before going on to recommend electroconvulsive therapy, a trial of zolpidem is probably in order as more and more states are tightening criteria for electroconvulsive therapy.

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LETTERS TO THE EDITOR SUBMISSIONS

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