The Use of Electroconvulsive Therapy in Atypical Psychotic Presentations: A Case Review

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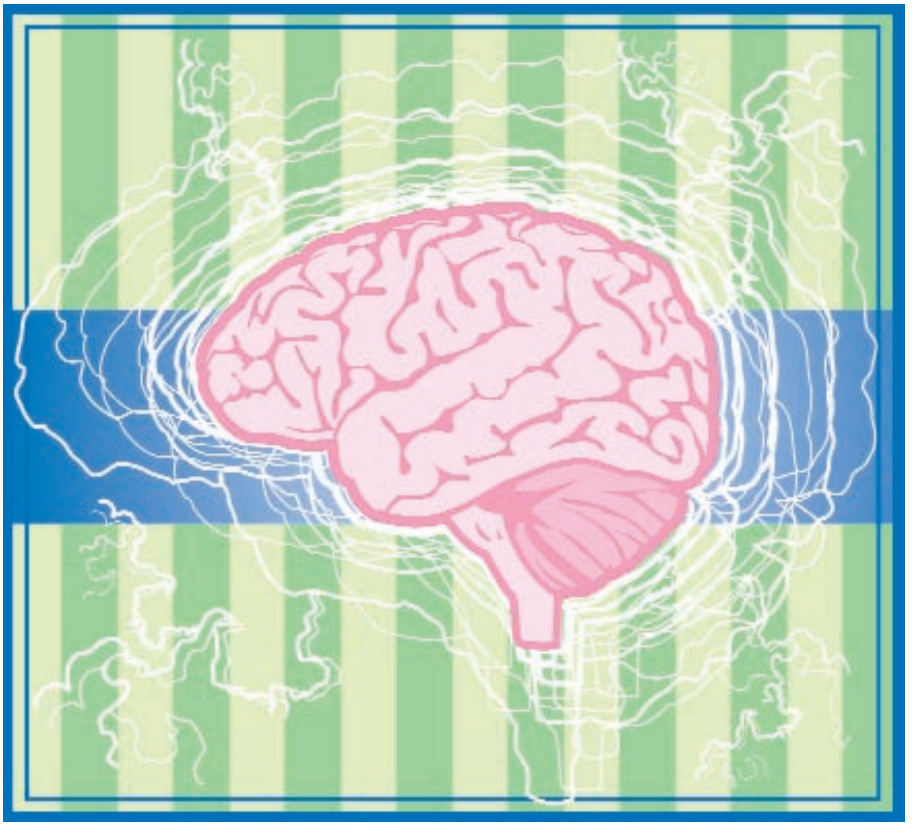
ABSTRACT

Convulsive therapy and its progeny, electroconvulsive therapy (ECT), were originally used for the treatment of catatonic schizophrenia, and there is little doubt that ECT remains an effective intervention for the treatment of schizophrenia. However, current practice tends to favor the use of ECT in severe or treatment refractory affective disorders, and its use in schizophrenia and other nonaffective (atypical) psychotic disorders has become controversial.

Case reports have suggested a role for ECT in two specific atypical psychotic disorders: Cotard's syndrome and cycloid psychosis. In this article, we review the atypical psychotic disorders and report a series of five case examples that signify the role of ECT in atypical psychotic presentations, particularly when the symptoms resemble those found in Cotard's syndrome and cycloid psychosis.



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INTRODUCTION

Convulsive therapy was designed with the treatment of schizophrenia

in mind, when in 1934, Hungarian neuropathologist Ladislas Meduna injected camphor-in-oil into a schizophrenic patient to treat catatonia.[1] The patient seized and survived, and Meduna repeated injections at 3- to 4-day intervals, and after the fifth injection, the patient became alert and talkative. After two additional seizures, the patient was no longer psychotic or catatonic. Despite being catatonic for four years, the patient returned to his community and began working again.

Meduna's pioneering work in convulsive therapy was rooted in an "antagonism hypothesis" that schizophrenia and epilepsy are diametrically opposing disorders. Noticing great differences in brain glial cell levels between epilepsy patients (increased) and patients with schizophrenia (decreased), Meduna studied the literature for evidence of similar antagonism between the two disorders.[1,2] He found reports of psychotic symptoms remitting when patients developed seizure disorders. He also noticed that the prevalence of epilepsy in patients with schizophrenia was rare, relative to its prevalence in the general population. These experiences encouraged Meduna to explore seizures as an antidote for schizophrenia.

Electroconvulsive therapy was introduced in the 1930s and proved to be a safer alternative to Meduna's method. In 1938, the Italian physicians Cerletti and Bini treated the first human with ECT—a delusional, incoherent patient who improved after one treatment and remitted after 11 treatments.[3] Although the first diagnostic indication for ECT was schizophrenia, modern practice has generally moved away from this. The vast majority of patients receiving ECT in recent years have primary affective disorders. Nonetheless, there remains a place for ECT for the treatment of schizophrenia and other psychotic disorders.

A few studies have tried to determine predictors for ECT response in schizophrenia. Although somewhat limited methodologically, these studies have shown the trends outlined in Table 1.[4]

TABLE 1. Clinical predictors for ECT in schizophrenia

FACTORS ASSOCIATED WITH FAVORABLE ECT OUTCOME	FACTORS ASSOCIATED WITH INFERIOR ECT OUTCOME
Catatonia	First rank or Schneiderian symptoms
Perplexity or confusional state	Premorbid Cluster A personality traits
Predominant positive symptoms	Presence of negative symptoms
Shorter duration of exacerbating episode	Chronically ill schizophrenics

In 2001, a task force of the American Psychiatric Association opined that ECT was effective for psychotic schizophrenic exacerbations of short duration, "…in the context of catatonia...when affective symtomatology is prominent, or when there is a history of favorable response to ECT."[5] However, the task force cautioned that even in these groups, the short-term benefits of ECT are likely to be equivalent to or less than those of monotherapy with antipsychotic therapy. The task force also reported that ECT was effective in the treatment of schizophrenia spectrum disorders, such as schizoaffective and schizophreniform disorders, and in atypical psychoses, "…when the clinical features were similar to those of other major diagnostic indications."[5]

ATYPICAL PSYCHOTIC DISORDERS

Atypical psychotic disorders are conditions that cannot be easily classified as either schizophrenia or a mood disorder with psychotic features. They comprise a widely varied and poorly understood collection of disorders and generally are divided into two subgroups according to duration of episode (transient versus persistent).[6]

Transient psychotic disorders. While schizophrenia and affective disorders have dominated the psychiatric literature and research efforts in psychotic disorders, several other types of psychotic conditions are emerging as significant. Included among these are psychotic disorders secondary to medical conditions or substance abuse, as well as a complex group of acute and brief psychotic disorders.[6]

Although the transient [atypical] psychotic disorders share basic pathological features of acute onset (<2 weeks), disturbances in reality testing, and bizarre or disorganized behavior, they vary widely in other respects, including geographic epidemiology, cultural framework, and descriptive symptomotology. Culturally and geographically, there is a disproportionately high frequency of transient psychotic disorders in developing countries of the Americas, Africa, and Asia.[7] Prominent concepts in the nosology of transient psychotic states include the *bouffées délirantes* described by French, the *reactive* or *psychogenic* psychoses and schizophreniform psychoses of

TABLE 2. Transient atypical psychotic disorders

SYNDROME	ONSET	PSYCHOTIC SYMPTOMS	AFFECTIVE SYMPTOMS	SENSORIUM	COURSE
Bouffées délirantes	Sudden (<48 hr) onset in persons w/o prior psych history	polymorphic delusions and hallucinations	Some associated emotional instability	Clouded consciousness, dissociative symptoms	Rapid return to premorbid level
Psychogenic [reactive] psychoses	Acute reactions to psychosocial stress or deprivation	paranoia, non-bizarre symptoms	Marked emotional distress and affective instability	Confusion	Few hours to several weeks duration
Schizophreniform psychoses	Sudden onset following a precipitating factor	Absence of negative symptoms	Disturbances of mood	Clouding of consciousness	Good prognosis, duration <6 months
Cycloid psychoses	Sudden onset not typically following a precipitating stressor	Mood-incongruent delusions, motility disturbances, ecstasy, blissfulness	Rapid mood swings (elation to depression), anxiety, preoccupation with death	Perplexity and confusion	Phasic course: Good immediate outcome but high recurrence

Scandinavian populations, the German concept of *cycloid* psychoses, and a number of culture-bound syndromes.[8] In the United States, nonaffective acute remitting psychoses are broadly categorized using the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision* (DSM-IV TR) terminology *brief psychotic disorder.*

Traditionally, European psychiatrists have utilized richer, more descriptive classification systems to characterize these psychotic states. Some of these disorders are summarized in Table 2.[6–8]

Persistent psychotic or delusional disorders. Like the acute or transient subgroup of atypical psychotic disorders, persistent psychotic or delusional disorders are regarded as neither schizophrenic nor affective illnesses and vary greatly from country to country. Included in this group are *folie á deux*, delusional jealousy, paraphrenia, Capgras syndrome, erotomania, and Cotard's syndrome. Some of the more commonly described persistent psychotic disorders are presented in Table 3.[6]

In DSM-IV TR, these concepts are captured in the diagnoses of delusional disorder and shared psychotic disorder and classified under the heading "Other Psychotic Disorders."[9]

CASE REPORTS

There are several published reports of successful treatment of the atypical psychotic disorder Cotard's syndrome with ECT.[10–12] There have also been case reports of ECT successfully treating Leonhard's, or cycloid, psychosis.[13–15] In this case series, we discuss five female patients admitted to Mississippi State Hospital with atypical psychotic presentations who were treated to remission with ECT.

Patient 1. A 44-year-old unmarried Caucasian woman with multiple previous psychiatric hospitalizations and diagnosis of schizoaffective disorder, bipolar type, presented to our facility. She was a Hurricane Katrina evacuee and was living in a group home prior to admission. She was supposed to be taking clozapine 200mg/day, but appeared to have either stopped or run out of this medicine.

Her past treatment history included lithium, valproic acid, chlorpromazine, thioridazine, haloperidol, fluphenazine, quetiapine, olanzapine, and risperidone. She had also been treated with ECT in 2001. Her mental health history was also notable for bulimia nervosa, polysubstance abuse, and recurrent suicide attempts. Her medical history was notable for chronic hypothyroidism.

The patient was a college graduate and a professionally trained dancer. She was not employed at the time of her admission and was receiving Social Security income. Her sister and mother were known to have been previously admitted to psychiatric facilities.

On mental status examination, this patient presented as confused, disheveled, and motorically agitated. She displayed odd, stereotyped behaviors, such as

TABLE 3. Persistent atypical psychotic disorders

SYNDROME	ONSET	PSYCHOTIC SYMPTOMS	AFFECTIVE SYMPTOMS	SENSORIUM	COURSE
Delusional disorder (syn. delusional jealousy, erotomania)	Can be sudden or gradual	Systematized, nonbizarre delusions that have been logically developed	Affective symptoms not typically seen	Behavior is typically organized, sensorium intact/alert	Stable, often chronic course
Shared psychotic disorder (syn. *Folie á deux*)	Development of delusions in context of close relationship with mentally ill person (primary case)	Delusions, hallucinations (rare) transferred from primarily (mentally ill) person to secondary person	Comorbid depression has been described	Generally alert, sensorium intact	Symptoms will remit if secondary person separated from primary case
Paraphrenia	Late age of onset	Well organized paranoid delusions (+/- hallucinations)	Not usually associated with affective disorders	Absence of dementia or confulsion	Absence of deterioration despite a protracted course
Capgras syndrome	Occurs over a wide age range and in a variety of comorbid illness states	Misidentification syndrome where familiar person believed to have been replaced by an imposter	Not usually associated with affective disorders	Generally clear, but dissociative symptoms reported	Variable
Cotard syndrome	Occurs over a wide age range, often in schizophrenia or severe depression	Nihilistic delusions that one has lost possessions, body, or does not exist	Usually associated with severe forms of depression	Can occur in the context of organic mental disorders	Three stages: germination, blooming, and chronic

tapping her hands rhythmically and counting under her breath. Her speech was rapid and pressured, and she displayed an odd, affected manner of speech reminiscent of a cockney British accent. Her thoughts were loosely organized and she exhibited clanging. Her affect was described as elated, labile, "giddy," and mood incongruent. She described her mood subjectively as "lonely and depressed." The patient exhibited multiple, bizarre delusions, which were of a violent nature (e.g., Charles Manson, serial killers, dismembered bodies).

Routine admission lab work was unremarkable. Thyroid studies were normal. Human immunodeficiency virus (HIV) and rapid plasma reagin (RPR) screens were negative. Weekly lab monitoring of blood cell count revealed a consistently elevated absolute neutrophil count (ANC) and white blood cells (WBC). Serum clozapine levels ranged from 328 to 707.

Hospital course. Throughout the first month of hospitalization, the patient remained clinically unchanged and often required emergency medications. Her behaviors included picking up non-food items off the floor and out of sink drains and eating them, inducing vomiting, and then mopping the vomit off the floor using her hair. She also appeared to have visual hallucinations, as evidenced by pointing out "witchcraft books" on the wall. Her speech was pressured and thought processes were loose. She was sleeping 3 to 4 hours per night, on average.

During Month 2, the patient was noted to be sleeping approximately six hours per night. Her speech appeared less pressured but she still displayed clanging and loosening of associations. Her hygiene remained poor. She ate very little and frequently induced vomiting. Her behavior remained grossly disorganized. She routinely vomited and spit on the floor and would wipe her hands through her own expectorated fluids.

During the course of her hospitalization, the patient's psychopharmacologic regimen

primarily consisted of clozapine and lithium. She was referred for ECT during her second month and underwent a series of eight acute treatments. Her ECT treatment course is outlined in Table 4.

Within two weeks of starting ECT, the patient's mental status rapidly improved. Her thoughts became more coherent and logical, and she exhibited a euthymic mood. She was eventually discharged to a group home in her community.

Patient 2. An unmarried 29-year-old African American woman was transferred from a regional crisis center, where she had been treated with a combination of clozapine and low-dose loxapine, to our facility.

The patient's first reported psychiatric treatment occurred at the age of 17 when she was treated for a depressive episode that began shortly after the death of her grandfather. She subsequently had multiple psychiatric hospitalizations. There is no known substance abuse history. Her medical history was notable for mild anemia and penicillin allergy. She graduated from high school and briefly attended college, but had no known work history.

On admission to our facility, the patient was grossly delusional, believing that she had 900 children by "immaculate conception." She appeared to have auditory hallucinations, as evidenced by laughing and talking to herself. Her thought processes were loose, with frequent use of non-sequitur language and neologisms. She was neither confused nor cognitively impaired. Although superficially pleasant, the patient was not very cooperative during initial assessment and appeared guarded.

Initial laboratory findings were unremarkable, with negative urine toxicology screen.

Hospital course. The patient was continued on clozapine 500mg in divided doses. She continued to be paranoid and evasive, and no clinical improvement was noted. She would frequently stay in her room all day and avoid contact with peers and staff. Clozapine dosage was increased. During the course of her hospital stay, she was started on isoniazid (INH) prophylaxis for a positive tuberculin skin test. Her clinical condition continued to worsen, with increased mood-incongruent auditory hallucinations and impairment in her language and communicating abilities. Her behavior became increasingly negativistic and hostile. Her attending physician became concerned that her deteriorating mental state could be iatrogenic, and INH therapy was discontinued. However, this intervention appeared to have no impact on her symptoms.

Over the course of her hospitalization, the patient was placed on several different medications, including risperidone, haloperidol, fluphenazine, and olanzapine, but she remained psychotic with disorganized thought processes, psychomotor retardation, akinesia, neologisms, hallucinations, delusions, paranoia, and intermittent agitation/hyperactivity. She was tried briefly on sertraline to target possible affective symptoms (crying, lack of participation, diminished appetite), and this appeared to have an activating, but not therapeutic, effect. Her speech became rapid, her affect became expansive, and she was noted to routinely sing in a loud voice and dance in the hallway. Around this time, her language and communication difficulties worsened to the point where the patient was unintelligible. At times, she would only communicate by spelling out words. She also began to speak in her own private language, which consisted primarily of neologisms and dramatically mispronounced words. The patient's behavior was unpredictable and bizarre. For example, during a pet therapy activity, she was noted to be completely withdrawn until the end

TABLE 4. ECT data on case examples

CASE SAMPLE	AXIS I DIAGNOSIS	# OF ACUTE TREATMENTS	LEAD PLACEMENT	PRE-ECT BPRS	POST-ECT BPRS
Patient 1	SAD-M/P	8	LA/RT(3) BT (5)	61	8
Patient 2	CUD	17	BT	53	5
Patient 3	SAD-Mx/P	8	LA/RT	44	15
Patient 4	MDD	14	LA/RT	60	26
Patient 5	BAD-D	6	LA/RT	47	6

SAD=Schizoaffective disorder, manic with psychotic features
CUD=Chronic undifferentiated schizophrenia
SAD-Mx/P=Schizoaffective disorder, mixed with psychotic features
MDD=Major depressive disorder
BAD=Bipolar affective disorder
BT=Bitemporal
LART=Left-anterior/Right-temporal
BPRS = Brief Psychiatric Rating Scale Score

of the group, at which time she ran over to the therapy dog and began speaking directly to it, using her private language. The patient's appearance also became more bizarre around this time, as evidenced by wrapping her head in rags and making adornments to her clothing out of garbage. Sertraline was decreased and she was started on valproic acid. She also underwent a trial of lorazepam due to her physician's clinical suspicion that her deteriorating behavior was a form of catatonia. However, these interventions had limited impact on her overall mental status.

Since the patient had not shown much improvement to a variety of pharmacological treatment approaches, ECT consultation was ordered. She went on to receive an acute series of 17 ECTs. After one week of treatment, the patient's language dramatically improved and she became more communicative with her treatment team. Other improvements noted early in her ECT course included euthymic mood, coherent speech, logical thought processes, and increased socialization. Her ECT course is summarized in Table 4. The patient was discharged to a local community mental health clinic with regular follow-up scheduled.

Patient 3. A 46-year-old unmarried African American woman had been admitted twice to a private psychiatric facility in her area within a four-month time period. Information supporting her court commitment stated that the patient believed for several weeks that her body was disappearing and was being held together by a string. While briefly rehospitalized in a private facility to await commitment, she was treated with citalopram, fluphenazine, olanzapine, clonazepam, and ziprasidone.

The patient was without any psychiatric history prior to age 46. In the year prior to her admission to our facility, she underwent a total hysterectomy. Records from her surgical follow-up indicated that she complained of feeling like she did not have a body. There was no known history of substance abuse. She denied any prior episodes of postpartum depression or psychosis, and there were no previous manic episodes described. Medical history was notable for rheumatoid arthritis, hypertension, and unspecified childhood head injury with possible loss of consciousness at two years of age.

The patient was never married and worked for several years as a manager of a motel. She had two grown children. She was the middle of seven children born to an intact family and denied any history of abuse. Family history was notable for a maternal aunt who was admitted to a state psychiatric facility.

Upon presentation to our facility, the patient was completely somatically preoccupied. She appeared to be very anxious and moderately distressed and volunteered, "My body is disappearing. My body is being destroyed. My body is gone." She appeared mildly sedated from her psychotropic regimen but was cognitively intact. She described her mood as "not good" and her affect was blunted. Other than her somatic delusion, there was no evidence of thought disorder or psychosis. On physical examination, she was morbidly obese and neurological examination was remarkable for some rigidity of tone in her cervical muscles.

Initial laboratory findings were suggestive of mild microcytic anemia, but otherwise unremarkable. An magnetic resonance imaging (MRI) of her brain suggested a possible type I Arnold-Chiari malformation.

Hospital course. During her initial hospitalization, the patient continuously obsessed about her body disappearing. She did not attend to her hygiene and often refused to eat or sleep. She was very restless, energetic, and talkative. Staff frequently found the patient staring into her bathroom mirror, insisting that her body was missing. Additional pharmacological management included quetiapine, pimozide, and clozapine, with no improvement in her symptoms.

Our staff neurologist evaluated the patient and opined that her possible brain malformation was not related to her mental status abnormalities and did not preclude her from being an ECT candidate. She was consulted for ECT and went on to receive a series of eight treatments. She demonstrated improvement in her somatic delusional system within one week of ECT, and after eight treatments, she verbalized no somatic complaints and there was no evidence of delusion.

Patient 4. A 48-year-old unmarried Caucasian woman with a long-standing history of mental illness, including five prior admissions to our facility, presented to our facility. Her latest admission was prompted by increasing paranoia about her neighbors, including the belief that she and her mother would be harmed. She reportedly had been "very depressed" after hurricane Katrina and her condition gradually deteriorated.

The patient's psychiatric history was notable for recurrent episodes of depression, and she was treated with venlafaxine, paroxetine, and mirtazepine. There were no prior manic episodes. Her prior diagnoses included major depressive disorder and personality disorder with borderline and dependent features. She also had remote alcohol and substance use history, with four prior admissions to a chemical dependency unit, but the patient had not used drugs or alcohol for the past eight years. Her medications at time of admission included trifluoperazine 5mg/day, quetiapine 100mg/day, and lorazepam 3mg/day. Previous manic episodes were denied by the patient.

There was a strong family history of mental illness. [Incidentally, her sister is Patient 1 in this case

series.] The patient graduated from college and briefly attended a graduate program in English. She had not worked for many years, and lived with her mother. Her legal history is remarkable for arrests for driving under the influence of drugs or alcohol (DUI) and public intoxication. The patient described herself as being chronically shy and unhappy, beginning in early childhood.

Upon presentation to our facility, the patient explained that she had recently left home to escape from her neighbors and believed that she would soon be killed. She described anhedonia, sleep disturbance (insomnia), and decreased appetite. She was preoccupied with somatic beliefs that she was rotting, malodorous, and that her brain was deteriorating. She described that her neighbors "…thought I was disgusting…I smelled bad like I do today," and seemed to believe that they were trying to poison her. The patient was also preoccupied with the belief that she was going to hell, and at one point during the interview suspected that she had already died and was living in hell. On mental status examination, the patient was cognitively intact except for mild impairment in her recent memory. Her affect was constricted and depressed, and she described her mood as, "It could be good, but I haven't slept or eaten well for days."

Initial laboratory findings were unremarkable, including a negative urine drug screen.

Hospital course. On Hospital Day 1, when a nurse tried to perform venipuncture, the patient refused, stating, "This is not a real situation…I will be brain dead tomorrow, so I just want to stay up and enjoy the night." Initial management consisted of treating her mood and psychotic symptoms with duloxetine, olanzapine, haloperidol, lorazepam, and valproic acid. Over the first several days, she routinely refused medications and meals and refused to leave her hospital bed. Her condition deteriorated to the point where she refused to drink anything and insisted that she was dying or even dead. She was referred to the ECT service since she had failed other interventions and was at immediate risk due to refusing to eat or drink. She received an acute series of 14 ECT treatments. Within two weeks of starting ECT, the patient showed marked improvement in her delusional system and modest improvement with depressive symptoms. She recalled believing that she was in hell earlier in her hospitalization and joked that, "Now I'm in purgatory."

Criteria for diagnosis of cycloid disorder include psychotic episodes of sudden onset, mostly unrelated to stress, with good immediate outcome but a high risk of recurrence, characterized by mood swings (from depression to elation) and at least two of the following: Perplexity or confusion; delusions of reference or persecution and/or mood incongruent hallucinations; motility disturbances; periods of ecstasy; and states of overwhelming anxiety.[13]

Patient 5. A 49-year-old divorced Caucasian woman with a history of bipolar disorder was admitted to a crisis stabilization unit (CSU) after attempting suicide by cutting her wrists. After two weeks at the CSU, she was court committed and transferred to our facility.

Up until several months ago, when the patient moved in with her father, she was living independently. She was the second of eight children. All of her siblings were treated for depression. Her mother was deceased secondary to lung cancer. The patient was reportedly molested by her father when she was a child. She was married and divorced twice and had two children, ages 21 and 13. Her highest level of education was two years of college, and she last worked in 1999 as a bank teller.

The patient's psychiatric history was remarkable for two prior admissions to local psychiatric facilities, and she was followed by her regional mental health center intermittently over the last 10 years. There was no known history of alcohol or substance abuse. Medical history was noncontributory.

Upon initial assessment, the patient described her first manic episode as occurring approximately 10 years previously, with elevated mood, hyperactivity, poor sleep, racing thoughts, and the need for hospitalization. She denied any previous episodes of major depression, but her history was suspicious for brief periods of mild depression. She also reported a recent history of auditory hallucinations and believing that people were breaking into her apartment, which led her to call 911 on a few occasions. She also reported a history of impulsive, parasuicidal behaviors, as evidenced by taking nonlethal overdoses in the context of abusive relationships. During the interview, she exhibited psychomotor retardation, speech latency, and made little eye contact. The patient did not exhibit any delusions or other evidence of psychosis. She demonstrated mild difficulties with recent memory, concentration, and abstract reasoning.

Initial laboratory findings were unremarkable, including a negative urine drug screen. Serum carbamazepine (CBZ) level on admission was 0.1, suggesting nonadherence with this medication. Subsequent CBZ levels were within expected therapeutic ranges.

Hospital course. Initial pharmacologic management consisted of risperidone, bupropion, trazodone, and carbamazepine. Within a few days of hospitalization, the patient began to reveal somatic delusions, including the belief that she had lost all control of her muscles and her body. Risperidone was increased, and she was administered a brief course of methyphenidate to treat her melancholic depressive features, but little improvement was noted. Around this time, it was noted that she was refusing to shower or bathe because she believed that she was "full of filth…it has gone too far."

During her second month of hospitalization, the patient remained passive and minimally interactive. She continued to exhibit somatic delusions, including the belief that she had lost control of her muscles and that she was "full of filth." Sertraline was added to her regimen, but when no improvement was noted, the patient was referred to the ECT service. Within one week of starting ECT, significant improvements were noted in the areas of her somatic delusions and depressive symptoms, and after six treatments, her somatic delusions had remitted.

DISCUSSION

Although, these case examples illustrate atypical psychotic presentations of relatively common psychiatric disorders, they share characteristics with distinct atypical psychotic disorders. Patient 1's predominant features of confusion, mood-incongruent delusions and hallucinations, mood lability, anxiety, and motility disturbances are consistent with cycloid psychosis. Patient 2 presented with nonaffective psychotic symptoms, including disorganized speech and thought processes and inappropriate affect, and went on to develop features of motility psychosis, catatonia, and cryptolalia, a phenomenon where the patient speaks in her own private language. Cases 3, 4, and 5 all share features of Cotard's syndrome, a delusional misidentification syndrome, whereby the patient believes that all or parts of his or her body are missing, or believes that he or she has died.

Cycloid psychosis is a transient, often recurring psychotic disorder, and is a diagnosis that is commonly used by European psychiatrists. The concept was introduced as "motility psychosis" by C. Wernicke in the early 1900s.[16] Wernicke's ideas were further developed by K. Kleist and K. Leonhard into the concept of cycloid psychosis.[17] Kleist reported two variants of the disorder: *confusional insanity* and *motility psychoses*.[17] Confusional insanity was characterized by contrasting phases of excitement and stupor, and motility psychosis was characterized by contrasting phases of hyperkinesia and akinesia. Leonhard went on to subdivide the disorder into "anxiety-elation," "confusion," and "motility" psychoses.[18] The concept of cycloid psychoses was updated by Perris in 1974, and criteria for diagnosis of the disorder were established.[13] These included psychotic episodes of sudden onset, mostly unrelated to stress, with good immediate outcome but a high risk of recurrence, characterized by mood swings (from depression to elation) and at least two of the following: perplexity or confusion; delusions of reference or persecution and/or mood incongruent hallucinations; motility disturbances; periods of ecstasy; and states of overwhelming anxiety.

Cycloid psychoses appear to be nosologically distinct from schizophrenia and schizoaffective disorder.[19,20] However, it is not uncommon for individuals presenting with cycloid psychoses to eventually be diagnosed with schizophrenia and schizoaffective disorder, despite having an excellent prognosis and high baseline functioning level.[21]

Cycloid psychoses have been reported to constitute a substantial part of psychotic disorder among women, particular in the postpartum phase.[22–24] Cycloid psychoses demonstrate a phasic course similar to bipolar disorders and generally have a good prognosis.[25] There are many parallels between delirious mania, excited catatonia, and cycloid psychoses.[26]

Treatment strategies generally target the predominant presenting symptoms, and antipsychotic medications are commonly used. There have been published reports of rapid, successful resolution of episodes of cycloid psychosis with ECT.[14,15]

Cycloid psychoses have been reported to constitute a substantial part of psychotic disorder among women, particular in the postpartum phase.[22–24] Cycloid psychoses demonstrate a phasic course similar to bipolar disorders and generally have a good prognosis.[25] There are many parallels between delirious mania, excited catatonia, and cycloid psychoses.

Although Patient 1 in the case series presented with many features of cycloid psychosis, she never experienced the complete remission often described in the course of the disorder. Patient 2 also lacked the expected complete interepisode remission, but the fluctuating pattern of akinesia and psychomotor agitation and prominence of mood-incongruent delusions and hallucinations were reminiscent of the disorder. Perhaps most interesting and unusual of Patient 2's presenting symptoms was the patient's cryptolalia, or development of her own private language. This

phenomena is occasionally seen in normal childhood language development (especially in twin siblings), and also can be found in pathological states, including Tourette's disorder, cerebral injury or ischemia, and schizophrenia.[27–29]

Delusional misidentification syndrome is an umbrella term for a group of delusional disorders that occur in the context of mental or neurological illness. These involve a belief that the identity of a person, object, or place has somehow changed or has been altered, and include Capgras, Fregoli's, and Cotard's syndromes. Cases of delusional misidentification have been presented as distinct syndromes, but in practice, they are variable and often occur simultaneously with other delusions, and in the context of other disease states.[30]

Cotard's syndrome was originally the term used to describe the delusion that one has died, does not exist, or who has suffered some other kind of catastrophic physical, socioeconomic, or existential loss.[6] The first case presented by Jules Cotard in 1880 was that of a 43-year-old woman who believed she had "no brain, nerves, chest, or entrails and was skin and bone…" and that she did not need food because "…she was eternal and would live forever."[31] Originally described as a form of affective illness, Cotard later referred to this clinical phenomenon as a delusional state *délire des négations* (nihilistic syndrome). Currently the term *Cotard's syndrome* is often limited to the belief that one is dead, which is overly restrictive.[31]

Cotard's syndrome is encountered in many disease states, including schizophrenia, severe depressive episodes, after trauma, during extreme hyperthermia consequent to infectious states, and during self-starvation.[32]

Treatment options for Cotard's syndrome are tailored to the underlying psychiatric or medical disorder. There have been several reports of successful resolution of Cotard's delusions with ECT, particularly in patients with underlying depressive disorders.

Patients 3, 4, and 5 in this case series presented with the classic features of Cotard's syndrome. The delusions of negation in Patients 4 and 5 occurred in the context of severe depression, whereas Patient 3 presented with prominent anxiety and hypomanic features. Patients 3 and 5 achieved full remission by the end of their ECT course. Patient 4 continued to have some residual depressive symptoms, which accounted for her post-treatment Brief Psychotic Rating Scale score (BPRS) score of 25. In each case, the use of ECT led to rapid improvement in the delusional system.

CONCLUSION

While some may remain dubious as to the nosological validity of atypical psychotic disorders, or how their presenting clinical features should be most logically and parsimoniously categorized, we feel that these disorders have clinical value, particularly when considering treatment strategies and prognostic implications. The role of ECT in schizophrenia and other nonaffective (atypical) psychotic disorders remains controversial, but some agreement exists about its usefulness in certain psychotic presentations. These case examples underscore the role of ECT in atypical psychotic presentations, particularly when the symptoms resemble those found in Cotard's syndrome and cycloid psychosis.

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