

## Case Report

# A Case of Tardive Akathisia Converting to Conversion Disorder

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## Abstract

We would like to present a unique case of the emergence of psychogenic akathisia, described as a conversion disorder, in a patient undergoing longstanding treatment for tardive akathisia. This is the case of a 54-year-old-female with generalized anxiety disorder, dysthymia, and dependent personality traits, complicated by a learning disability and prior alcoholism, with past episodes of psychosis. First hospitalized at 25, she experienced akathisia on numerous trials of typical neuroleptics. Subsequent psychotic episodes necessitated the re-addition of low-dose chlorpromazine but akathisia did not recur. However, tardive akathisia emerged twenty years later, and through joint treatment by neurology and psychiatry, she was eventually maintained on clozapine and clonazepam after several unsuccessful medication trials. These medications gave some relief but she retained a severe decline in her functioning. Eight years later, when admitted for a trial of ECT, the neurology consult team determined her symptoms were no longer consistent with tardive akathisia, but rather consistent with a psychogenic movement disorder. Clozapine was slowly discontinued and management of her anxiety became the focus of treatment. The patient was left with residual periodic akathetic-like movements and was given a new diagnosis of psychogenic akathisia. In the months since discharge, her abnormal movements have decreased while her social functioning has improved.

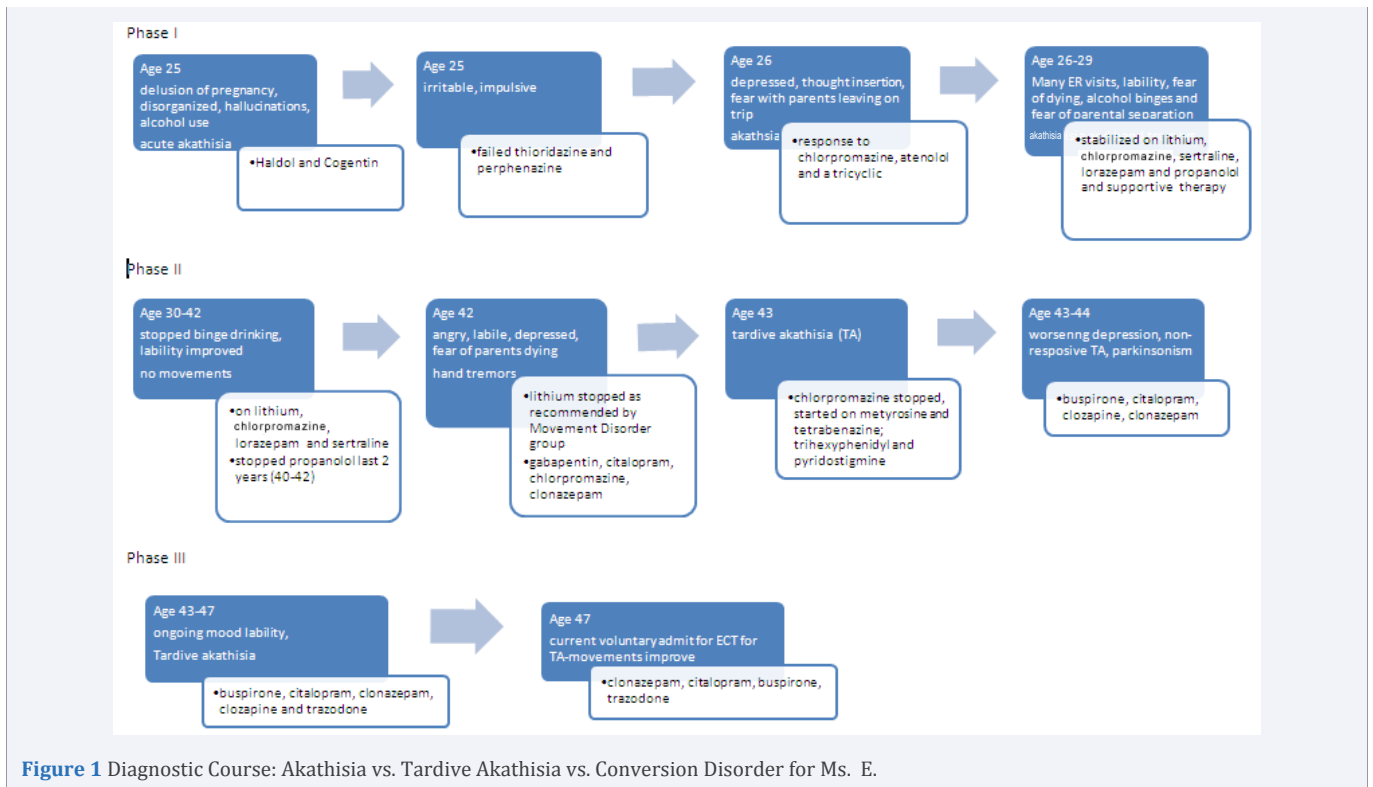
## CASE PRESENTATION

"Ms. E" is a 54-year-old woman referred to our clinic 29 years ago for outpatient treatment of atypical psychosis whose case we believe to be unique. She currently lives with her mother and her father stays with them 6 months a year (the other 6 months he lives in their country of origin). Ms. E is single and childless and has a married older brother. She completed high school but is unemployed, and is supported by disability payments. Since her first presentation to our clinic, she has had several hospitalizations and was given various diagnoses despite twice undergoing a battery of psychological tests, receiving episodic supportive therapy, and having had multiple medication trials. Her course of treatment is not atypical for many patients in public health care systems with multiple co-morbidities. Ms. E presented with a request to be voluntarily admitted to hospital for electroconvulsive therapy in an attempt to ameliorate her treatment-resistant akathetic movements. At the time of this request, she was in outpatient psychiatric treatment for generalized anxiety disorder and receiving specialized neurological care for tardive akathisia at our Movement Disorders Clinic. We present in great detail the relationship between medication trials and her movement disorders, and describe evolving symptoms. It is the retrospective review of this detailed

history and video recordings that resulted in the unexpected diagnosis of conversion disorder, attained in collaboration with both neurology and psychiatry.

## Phase I

**Please refer to figure 1 for medication timelines:** Ms. E was first hospitalized at age 25. At that time, she was feeling anxious about receiving a raise and a promotion at work without having earned a college degree. She also believed that she should be married and living outside of her parental home. At a party, Ms. E engaged in binge drinking and had to be taken home by her friends. Over the course of the next four days she did not sleep, urinated on herself, had psychomotor agitation in the form of pacing back and forth, and was under the delusion she was pregnant and about to give birth. She was brought to the emergency room by her parents where she thought the staff was trying to kill her. She endorsed auditory hallucinations of a female voice telling her "you should be married" and "you should move out," as well as thought insertion by her parents. Of note, toxicology, physical exam, and her electroencephalograph were all negative. Ms. E was admitted to the inpatient psychiatry unit and eventually given a diagnosis of generalized anxiety disorder and atypical psychosis with infantile personality disorder. The



**Figure 1** Diagnostic Course: Akathisia vs. Tardive Akathisia vs. Conversion Disorder for Ms. E.

typical neuroleptic haloperidol was started and the anticholinergic benztropine added to treat concurrent acute akathisia (Table 1).

Akathisia at this time was documented as “inability to remain seated”. Ms. E’s thought disorder resolved but she became rude, impatient, preservative, and unable to sit in groups and demanded to leave the hospital early. Within 5 months of hospitalization, she had short trials each of perphenazine and thioridazine, but her akathisia persisted and she developed a mild bilateral tremor in her hands. Concurrently, she started to exhibit a depressed mood and was then diagnosed with dysthymia. One month later, Ms. E became upset with her parents’ plan to leave for vacation and exhibited crying spells, insomnia, and the belief that her family members were saying she was a “bad person”. Her therapist determined that Ms. E had not been psychotic but rather was reacting to her parents leaving without her. Neuropsychiatric testing was conducted and the Wechsler Adult Intelligence Scale, the Bender Gestalt, Rorschach and Thematic Apperception Test were administered. The testing revealed she had borderline intelligence (full scale IQ 85) and “a self-concept of helplessness and ineffectiveness and fixation at prepubertal developmental levels [and...] unresolved dependency needs”. The patient improved with supportive therapy, although felt guilty when her mother decided to stay home with her. She then had a trial of a fourth neuroleptic, chlorpromazine, and for her continued akathisia, the addition of atenolol. A tricyclic antidepressant was briefly added to her regimen for emerging symptoms of depression.

One year later, she tolerated the discontinuation of chlorpromazine which also resulted in the cessation of akathisia. However, Ms. E then started a new job and became ill again

with extreme crying spells and the belief that her parents were “manipulating” her. A low dose of chlorpromazine was restarted and stopped after five months without recurrent akathisia. Three years later, chlorpromazine was restarted when after four days of binge drinking, Ms. E experienced insomnia just before she was to leave on vacation with her boyfriend. Over the next two years, Ms. E would have episodes of a fear of dying and lability of mood after bingeing on alcohol. These were somewhat relieved with the addition of lithium. Her medication regimen at this point was sertraline, lithium, lorazepam, and chlorpromazine. Of note, she continued to not have any akathisia on chlorpromazine. However, she did have the re-emergence of a bilateral fine hand tremor and so the beta blocker propranolol was prescribed. She remained on this regimen for the next twelve years until she agreed to stop propranolol. She remained off propranolol with no movement abnormalities for about two years.

## Phase II

Ms. E stopped drinking alcohol in her early forties which greatly improved her mood lability. She was referred to our Movement Disorders Clinic two years after discontinuing propranolol with complaints of the worsening of her bilateral hand tremor. A consulting neurological specialist in movement disorders recommended her lithium be discontinued; her tremor resolved but she became angry and depressed when her psychiatrist went on vacation. Gabapentin was added to decrease her re-emergent lability, and she reported a brief period of improvement. Ms. E was hospitalized three months later with complaints of depression and worthlessness, preservative thoughts about her parents dying and being left alone. An SSRI, citalopram, was started and she was given a diagnosis of mood

**Table 1:** DSM 5 review [1].

Acute Akathisia	Tardive Akathisia
“Subjective complaints of restlessness often accompanied by observed excessive movements...developing within a few weeks of starting or raising the dosage of a medication...or after reducing the dosage of a medication used to treat extrapyramidal symptoms.”	“Tardive syndrome involving other types of movement problems, such as dystonia or akathisia, which are distinguished by their late emergence in the course of treatment and their potential persistence for months to years, even in the face of neuroleptic discontinuation or dosage reduction.”

**Table 2:** Diagnostic Course: Akathisia vs. Tardive Akathisia vs. Conversion Disorder for Ms. E.

Symptoms and Signs	Akathisia- Phase I	Tardive Akathisia (TA) - Phase II	Conversion Disorder- Phase III
Inability to remain seated	X	X	X
Legs shaking when seated		X	X
Marching in place when seated and standing		X	X
Crossing and uncrossing legs when seated		X	X
Movements disappear when distracted (during interview and with daily activities)			X
Evidence of movements particularly when examined / suggestibility			X
Unconscious gain= parents stay with her			X

**Table 3:** Review definitions: Psychogenic Movement Disorder *is like* Conversion Disorder.

Psychogenic movement disorder (akathisia) IS	Like Conversion Disorder
Psychogenic movement disorder is a clinical syndrome defined as the occurrence of abnormal movements that result from a psychiatric cause rather than a general medical or neurologic cause (8)	Conversion disorder (functional neurologic symptom disorder) is characterized by neurologic symptoms (eg, weakness, abnormal movements, or nonepileptic seizures) that are inconsistent with a neurologic disease, but nevertheless cause distress and/or psychosocial impairment (1)

disorder not otherwise specified. At this time she was taking gabapentin, citalopram, clonazepam and chlorpromazine.

Ms. E then developed movements that included the shaking of her legs, marching in place, crossing and uncrossing her legs, and an inability to sit still, finding relief only in sleep. A retrospective Barnes scale (BARS) would have been scored at approximately 13-14 points total [2]. Ms. E was extremely dysphoric due to her discomfort, and was diagnosed by her neurologist at the Movement Disorders Clinic with tardive akathisia (Table 1). She was hospitalized emergently to slowly titrate off the chlorpromazine and start metyrosine. During the month in hospital, tetrabenazine was added to the metyrosine with little improvement and several side effects, including worsening of depression and severe Parkinsonism. As she continued to have medication-resistant tardive akathisia for several months post hospitalization, ECT was recommended. Ms. E refused ECT, and after obtaining a second neurologic opinion, a trial of clozapine was started. This medication allowed for the taper and discontinuation of metyrosine and tetrabenazine, as well as trihexyphenidyl and pyridostigmine, which were used to try to control side effects.

### Phase III

Unfortunately, Ms. E's akathisia and functioning continued to worsen and she was hospitalized three years later for delirium after confusing her medications and taking incorrect dosages. Neuropsychiatric testing was repeated and she was administered the Wechsler Adult Intelligence Scale IV, Millon Clinical Multiaxial Inventory III, Woodcock-Johnson Tests of Achievement and

the Adaptive Behavior Assessment System II, among others. She was again found to have borderline intelligence (full scale IQ 76), a decline in her working memory compared to her prior results, a primary affective illness and again, “dependent [...] with a fear of abandonment”. A brain scan (computed tomography) found no abnormalities. Surprisingly, her akathisia improved, though did not abate, while she was in hospital. It was suspected then that her tardive akathisia had improved by virtue of administration of timely and correct doses of all her medications, especially clozapine. Ms. E spent the next four years on buspirone, citalopram, clonazepam, clozapine and trazodone, now pre-poured in pill boxes for her, with fair improvement in her symptoms but an inability to attend the clinic rehabilitative program or socialize. She continued to be followed by neurologists at the Movement Disorders Clinic for treatment of her tardive akathisia. These neurologists had videotaped her movements from the beginning of her care there.

Ms. E, her family, and her outpatient psychiatry and neurology teams then decided to reconsider a trial of ECT for tardive akathisia. However, her akathetic movements quickly resolved again after she was admitted to the inpatient unit. She was evaluated there by both her outpatient neurology and psychiatry providers who concurred that there was a marked improvement of her movement disorder. Further observation revealed that Ms. E displayed leg-shaking movements (tapping the heel on the floor repetitively while keeping the toes on the ground) when she was interviewed and examined. However, casual observation in the inpatient unit showed that she exhibited no abnormal movements during her routine activities. She was observed to

sit watching television completely still for several minutes at a time without moving her legs, shifting position, getting up, or marching in place. Additional examination also revealed that her leg-shaking movements, which appeared within minutes of starting an examination, reliably disappeared with distraction maneuvers, and at times appeared following suggestion.

A hospital-wide case conference was held, her videos were reviewed, and the consensus was that her movements no longer met criteria for tardive akathisia and were possibly psychogenic in origin. The decision was then made to discontinue her clozapine, prescribed solely for tardive akathisia, and monitor closely for re-emergence of a movement disorder. There was no worsening or re-emergence of involuntary or abnormal movements. The appearance of her movements appeared to be more closely linked to parental separations and her inability to express her distress about separations. This was reminiscent of her reaction in Phase I to her parents' planned vacation, or her reaction in Phase II to her psychiatrist's vacation. Ms. E's functioning has greatly improved since this last hospitalization. When experiencing stress, Ms. E will once again march in place or complain of the fear of her parents dying; however, she can calm herself down with learned cognitive behavioral techniques. A recent BARS score was a total of four points. Ms. E is attending clinic weekly and resuming a more active social routine.

## DISCUSSION

Tardive akathisia has a variable incidence rate of 0.1%-41% in the literature (Table II) [3]. Ms. E's risk factors for developing tardive akathisia include her age, gender, cognitive impairment, a history of alcoholism, affective disorder, and prior akathisia [3,6,7,9]. In Ms. E's case, we were fortunate to have documentation of her entire 29 year history of treatment. Review of video and discussions with prior treating neurologists confirmed that Ms. E did indeed have tardive akathisia in the past, which emerged well after 3 months of treatment with chlorpromazine, a neuroleptic widely noted in the literature as causing this syndrome [6,9,10,12]. Interestingly, more recent literature suggests that the discontinuation of lithium could precipitate akathisia, since in rare cases it can cause tardive syndromes [4]. It is plausible that she may have already had some resolution of her tardive akathisia and the development of a psychogenic akathisia during her penultimate hospitalization, given that the symptoms abated with distraction maneuvers and partially resolved in hospital except in times of stress [7,8]. It is important here to note that the use of ECT as a treatment for tardive akathisia remains controversial [5,9,11].

Currently, Ms. E's symptoms are reproducible when discussing her parents leaving, either literally when on vacation or metaphorically when imagining their inevitable demise. After extensive discussion with our Movement Disorders specialists, our hypothesis is that the tardive akathisia, an involuntary movement disorder, has developed into a conversion disorder with the psychological stressor of separation; that is, a movement disorder with psychogenic etiology, as there is an unconscious secondary gain to reproducing the same behavior [7]. Ms. E is not intentionally producing the movement disorder, as would be the case in a factitious disorder. We noted Ms. E received considerable attention from this behavior such as her mother's

decision to stay home with her instead of leaving for vacation with her father. Ms. E and her mother have an interdependent relationship where both benefit from her infantile behavior. A psychogenic component also explains the marked improvement she showed during the most recent hospitalization where she received several cognitive coping tools.

One could argue that the tardive akathisia is still being treated by benzodiazepines and may not be visible on examination. However, we believe this to be unlikely because Ms. E has developed symptomatology that no longer meets criteria for tardive akathisia. Her movements are not specific to akathisia and fluctuate between being absent to steadily present over the course of minutes. Fluctuations due to anxiety cannot explain the qualitative change in the nature of the movements either. Again, her symptomatology is strongly suggestive of a psychogenic movement disorder, in that it appears in highly specific context and is sensitive to suggestion and distraction [6,7,9-11].

## CONCLUSION

We have been unable to find in the literature another case of tardive akathisia resolving and then apparently converting to conversion disorder, or more specifically in this case, psychogenic akathisia (Table 3).

Tardive syndromes present a very complex diagnostic challenge for which a team effort is needed of both neurology and psychiatry [7]. We believe that Ms. E's development of a conversion disorder is unique in the literature, and hope it will encourage clinicians to continually re-evaluate diagnosis and medications. This is especially important if patients exhibit ongoing distress and functional impairment, even if it seems that all treatment possibilities have been exhausted.

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