Prior to hospital admission the patient lost weight and had a viral-like illness which caused vomiting and she was at particularly high risk for complications such as severe electrolyte disturbance and its sequelae. It is clear that the cause of the development of central pontine myelinolysis was most likely secondary to rapid correction of her electrolytes, which had originally been acknowledged but not effectively communicated to the general medical team from Intensive Therapy Unit. Better awareness of this in the hospital staff could be facilitated by education about the risks of refeeding syndrome by liaison psychiatrists.

The gradual change in the patient's presentation (as is the case in central pontine myelinolysis), and her previous history of anorexia nervosa along with an initial normal MRI, made the pursuit of an organic cause less appealing. There was an overlap in symptomology of a neurological or psychiatric origin making diagnosis difficult, and so the patient was thought to be purposefully behaving in a manner that eventually alerted her to the attention of the liaison psychiatric services.

Attribution theory from the field of social psychology, as first described by Heider,[12] can explain the subsequent course that was seen. Once it was "believed" to be a functional problem, the attitude of staff was altered such they exhibited a "fundamental attribution error"—an unjustified tendency to assume that a person's actions depend on what "kind" of person he or she is rather than on the social and environmental forces at work. This not only delayed the diagnosis but also, to some extent, affected the initial psychiatric assessment with an acceptance of a functional problem and consideration of the use of the Mental Health Act.

Conclusion

As in similar case reports, we illustrate the potential pitfalls in the prevention, diagnosis, and management of the complications of refeeding syndrome in patients with mental illness who are malnourished as a consequence. We have also tried to demonstrate that to deliver effective liaison psychiatry services to a general hospital, one should be thorough when reviewing medical notes, communicate with all relevant professionals, and obtain collaborative history when possible from all sources, including the family and friends of the patient. One should also be aware that the stigma of mental illness will need to be counteracted to some degree with education, and how this stigma can also indirectly influence even our own clinical judgments.

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Psychosis in a Case of Schizophrenia and Parkinson's Disease

To the Editor: The dopamine hypothesis of schizophrenia states that increased dopamine activity is the cause of the productive symptoms in schizophrenia. Parkinson's disease in contrast is characterized by the loss of dopamine in the mesostriatal and mesolimbic system. Nuclear medicine provides two helpful methods in imaging the dopaminergic system: presynaptic dopamine transporter imaging with cocaine analogs (e.g., FP-CIT SPECT) and postsynaptic receptor imaging with D2 receptor ligands (e.g., IBZM SPECT). In idiopathic Parkinson's disease, typically only presynaptic transporter function is impaired. Only a few case reports confirmed in vivo by FP-CIT SPECT

that the coexistence of schizophrenia and Parkinson's disease can occur.[1,2] In addition to these reports we refer to treatment options of acute psychosis in schizophrenia and concomitant advanced Parkinson's disease with motor fluctuations.

Case Report

We report on a 74-year-old man with no dementia who was previously diagnosed with paranoid schizophrenia and repeatedly treated with first and second generation antipsychotics. Ten years ago parkinsonism was first described and antipsychotic treatment was switched to clozapine (62.5 mg/day). As the patient refused to take clozapine in the course of the disease it was replaced by olanzapine (5 mg/day). Additionally he received L-Dopa (400 mg/day) and pramipexole (4 mg/day) to treat parkinsonism. He responded well to treatment but later developed on/off-fluctuations and peak-dose dyskinesias, wherefore a treatment with amantadine (200 mg/day) was initiated. He then rapidly developed severe delusions. On admission we saw a psychotic patient who was disabled by severe on/off-fluctuation and peak-dose dyskinesias. As there was some doubt concerning the diagnosis of pure Parkinson's disease, FP-CIT SPECT was performed and it showed bilaterally marked reduction of striatal dopamine transporter binding in the putamen (Figure 1). Pramipexole and amantadine were stopped and quetiapine (125 mg/day) was added to the drug regimen, which led to marked improvement of the mental state. However, motor symptoms became worse. We increased the total dose of L-Dopa to 500 mg in combination with entacapone and shortened the intervals between the doses (five times 100 mg, every 3–4 hours) whereby good motor control was achieved.

Discussion

FP-CIT SPECT allows differentiation between drug-induced parkinsonism and Parkinson's disease.[3] As all antiparkinsonian agents can deteriorate the mental state, it seems useful to confirm the clinical diagnosis by means of a FP-CIT SPECT. There is an ongoing discussion about how extended medication contributes to the development of psychosis in Parkinson's disease. Some reviews recommend the reduction of antiparkinsonian drugs,[4] especially amantadine, anticholinergics, and dopamine agonists; others state that factors like dementia, age, or duration of disease contribute to the genesis of delusion and hallucinations.[5] In our case, the reduction of amantadine and pramipexole led to marked improvement of the delusions. This points to a relationship between medication and psychosis. We introduced quetiapine when the patient refused the clozapine treatment. We achieved good motor control by giving L-Dopa in shorter intervals and in combination with entacapone. This case illustrates the challenge of advanced Parkinson's disease in chronic schizophrenia. If a coincidence of these two entities is suspected, we recommend differentiating between drug-induced parkinsonism and idiopathic Parkinson's disease by presynaptic nuclear imaging.

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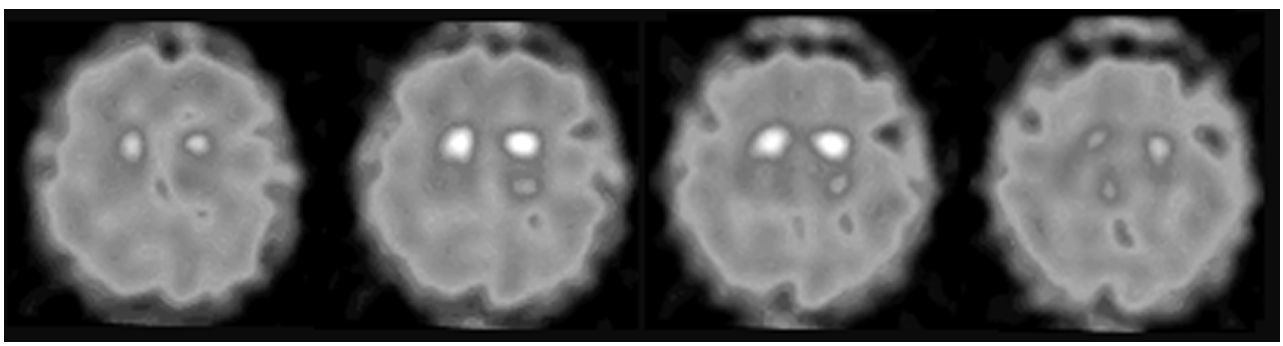
FIGURE 1. Presynaptic Dopamine Transporter Imaging with 123I-FP-CIT Reveals Markedly Impaired DAT Activity in the Lentiform Nucleus



There is some activity preserved in the caudate nucleus.

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Risperidone Treatment of Sleep Disturbances in Tourette's Syndrome

To the Editor: In 1885, Georges Gilles de la Tourette described a nervous disease characterized by lack of motor coordination, echolalia, and coprolalia.[1] Later, the disease became known as Gilles de la Tourette's syndrome and includes both verbal and motor tics with onset before the age of 18. Verbal tics may be simple (e.g., meaningless fonatory sounds, throat clearing, barking, etc.) or, less commonly, complex (coprolalia, echolalia, etc.), while motor tics are sudden, fast, repetitive, nonrythmic, stereotyped involuntary movements which may include blinking, facial grimacing, jumping, sniffing, or echopraxia.

According to Jankovic and Rohaidy,[2] sleep disturbances accompany Tourette's syndrome in 60% of cases. Nevertheless, no particular sleep disorder has been identified for this disease, although rhythmic periodic movements,[3] REM sleep behavior disorder [4] and parasomnias [5] are commonly described. Concerning sleep architecture, only a few polysomnographic studies have been reported with controversial results. Only in a recent well-controlled study, Cohrs et al.[6] found difficulties initiating and maintaining sleep, reduced sleep efficiency, an increase in stage 1 sleep, multiple arousals, as well as a decrease in slow-wave sleep.

Standard pharmacological treatment for Tourette's syndrome includes antidopaminergic drugs. Silay and Jankovic, in a more recent review,[7] stated that along with behavioral interventions, haloperidol and pimozide (dopaminergic antagonists) are the only two pharmacological agents approved by the Food and Drug Administration for the treatment of Tourette's syndrome.

In view of the fact that blockade of dopaminergic systems may in itself induce sleep disturbances,[8] we decided to analyze the sleep pattern of a 12-year-old patient with Tourette's syndrome before and after treatment with the antidopaminergic agent risperidone, an atypical antipsychotic previously reported effective in the treatment of Tourette's syndrome.[9,10]

Methods

A 12-year-old boy with no previous psychopharmacological treatment was diagnosed as having Tourette's syndrome, according to DSM-IV criteria. Following medical and neuropsychological evaluation, signs of attention deficit disorder and depression (i.e., anhedonia and hopelessness) were found.

EEG Recording

The patient was studied in the Sleep Disorder Center of the Universidad Autonoma Metropolitana in Mexico City. To discard additional EEG abnormalities, a 10/20 head mount was installed along with standard polysomnography recordings, including facial and limb electrodes, respiratory bands, and a microphone to record snoring and vocalizations. The patient was videotaped throughout the night. The study initiated at 10 p.m. and ended at 7 a.m. the next day. Sleep

TABLE 1. Sleep Characteristics of a Patient with Tourette's Syndrome Treated with Risperidone

	Before Treatment	After Treatment	Comparison Subjects[1] (Mean ± SD)
Total study time (min)	480	484	
Total sleep time (min)	454.5	474.5	523.7 (39.9)
Sleep latency (min)	20.5	8	11.7 (10.1)
REM latency (min)	102.5	105.5	117.9 (43.3)
Wake time (%)	4.2	1.9	4.1 (2.2)
Light sleep time (%)	47.9	64.6	57.9 (6.1)
Slow wave time (%)	36.6	21.7	22.2 (5.5)
REM time (%)	14.7	13.5	17.9 (4.1)
Arousals	183	32	25.4 (9.6)
Awakenings	8	4	4.1 (2.2)
NREM movs / hour	13.9	6	
REM movs / hour	28	6.7	

[1]Normal values reported for the age and sex of the patient. Data taken from Roberts JJ, Hoffman RF, Emslie GJ, et al: Sex and age differences in sleep macroarchitecture in childhoos and adolexcent depression. Sleep 2006; 29:351–358