Psychotic Symptoms and a Diagnosis of Schizophrenia Follow an Initial Diagnosis of Tic Disorder

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We report a patient with a tic disorder who later developed psychotic symptoms and was diagnosed with schizophrenia.

Key Words: tic disorder, schizophrenia, auditory hallucination, social dysfunction, haloperidol

INTRODUCTION

Gilles de la Tourette's syndrome and related tic disorders have been reported with comorbid psychological disorders such as obsessive–compulsive disorder (Frankel and others 1986; Pauls and others 1986; Singer and Walkup 1991) and attention-deficit hyperactivity disorder (Nee and others 1980; Comings and Comings 1987; Singer and Walkup 1991).

Comorbid schizophrenia has been reported to be quite rare (Shapiro and others 1972). Kerbeshian and Burd (1987) reviewed 11 cases of tic disorder in patients who presented with symptoms resembling schizophrenia and concluded that they did not meet the diagnostic criteria for schizophrenia.

We report the case of a patient with a long history of haloperidol treatment for a tic disorder who developed psychotic symptoms and was diagnosed with schizophrenia at the age of 16.

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CASE REPORT

The patient was a right-handed, 16-year-old woman. Her mother was hospitalized twice for threatened abortion during her pregnancy. The patient was diagnosed with congenital dislocation of the hip after birth and was treated in Tokyo Metropolitan Bokuto Hospital. Her developmental milestones were normal. Her family had no history of movement disorder or psychiatric disease. Facial twitching was 1st noticed by a pediatrician at the age of 5, and she gradually developed various motor tics. At the age of 9, she was 1st admitted to the Department of Psychiatry at the Tokyo Metropolitan Bokuto Hospital. Motor tics such as eye blinking, facial twitching, head shaking, shoulder jerking, and foot tapping were observed. No vocal tic was observed. The patient also displayed tendencies toward hyperactivity and impulsivity and was diagnosed with tic disorder. She was treated until the age of 15 with haloperidol up to 4.5 mg/d. She also participated in a series of play therapies for about a year. Her motor tics improved with those treatments, although she suffered occasional lapses.

She was admitted to the Department of Neuropsychiatry of the Tokyo Medical and Dental University at the age of 16, after having stopped taking medication for 13 mo. She was suffering from auditory hallucinations, which were manifest as a running commentary on her thoughts and behavior. She also presented with thought withdrawal and motor tics such as facial twitching and shoulder jerking. Additionally, her family reported that the patient made fantastic statements. No abnormality was found on physical examination, computed tomography scans, or electroencephalography. Treatment with haloperidol was restarted at 2 mg/d, then raised to 4 mg/d. Her psychotic symptoms and motor tics were partly reduced with this treatment, but her drug compliance was low, and she gradually avoided medication.

Fifteen weeks after the 1st admission, her hallucinations increased and she became excited: she was hospitalized in Onda Daini Hospital. Maximum doses of 30 mg of haloperidol, 400 mg of chlorpromazine, 30 mg of nemonapride, and 50 mg of levomepromazine were administered daily. She remained hospitalized because of her psychotic symptoms (both negative and positive) associated with marked social dysfunction 1 y after her 1st admission.

DISCUSSION

Our patient met DSM-IV (American Psychiatric Association 1994) criteria for chronic motor or vocal tic disorder when she was 1st admitted to the Department of Neuropsychiatry at the Tokyo Medical and Dental University. She had been diagnosed with Gilles de la Tourette's syndrome in Bokuto Hospital, but since no vocal tic was observed at any time, her symptoms did not meet the DSM-IV criteria for Tourette's disorder. Six months later, she met the DSM-IV criteria for schizophrenia.

She had no experience of drug abuse. In retrospect, Wilson's disease can be ruled out since she showed no neurological signs such as flapping tremor, rigidity, or speech disturbance, and Kayser-Fleischer ring was not observed.

Dopaminergic hyperactivity in tic disorders has been suggested since dopaminergic receptor (D_2) blockers like haloperidol suppress tics (Seigno 1961; Singer and Walkup 1991). At the same time, dopaminergic hyperactivity in schizophrenia is a well-known hypothesis. The present case, which met the DSM-IV criteria for both tic disorder and schizophrenia, suggests the possibility that a common biological mechanism of hyperactivity in the dopaminergic system may be involved in the development of both disorders.

Silva and others (1993) reported the case of a patient with Tourette's syndrome who developed transient neuroleptic-withdrawal psychosis. Yamamoto and others (1995) reported another case. Because our patient developed auditory hallucinations and other psychotic symptoms after she stopped taking haloperidol, the possibility of withdrawal psychosis exists. Silva and others reported that their patient's auditory hallucinations subsided with readministration of neuroleptics in a short period of time, but in our case, the patient's psychiatric symptoms continued. We do not think that withdrawal psychosis was likely, therefore, though the withdrawal of haloperidol may have acted as a trigger for the development of schizophrenia in this case. Postsynaptic dopaminergic supersensitivity after long-term administration of neuroleptics has been suggested by studies of postmortem brain tissue of patients with schizophrenia (Lee and others 1978; Owen and others 1978) and tardive dyskinesia (Jacobson and others 1974). Because the subject in our case study had been treated with haloperidol for years before her onset of schizophrenic symptoms, postsynaptic dopaminergic supersensitivity may have occurred. We do not think that the patient's persistent hallucinations or refractory reduction of psychosis is atypical of schizophrenia. Postsynaptic supersensitivity, however, might have played a role in its development.

Despite our assumption that a common biological mechanism may be involved in the development of both disorders, there remains a possibility that the 2 disorders coexisted by chance in our case.

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