Case Report

Misidentification of Wilson Disease as Schizophrenia (1998–2013): Case Report and Review

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ABSTRACT

Wilson’s disease (WD) is a neurodegenerative disorder due to copper metabolism. Schizophrenia-like psychosis and delusional disorder are rare forms of psychiatric manifestations of WD. The lack of recognition of these signs and symptoms as being attributable to WD often leads to delays in diagnosis and management. Knowledge about relationship of the psychiatric manifestations to WD can help with the administration of adequate management aimed at both the psychiatric issues and underlying WD. The objectives of this article are to review case reports whose subject is the incorrect diagnosis of schizophrenia or schizophrenia-like syndrome in patients with WD and to detail one case of this mismanagement of the disease. A 35-year-old unmarried Iranian woman presented to the consulting psychiatrist in the emergency room after a suicide attempt due to commanding auditory hallucination. She had previous eleven admissions in psychiatric hospital with major depressive episode with psychotic features, schizoaffective disorders, and then schizophrenia diagnosis. Nineteen years after her first symptoms, it was discovered that the patient was suffering from WD. We searched Google Scholar, Ovid, PsycINFO, CINHAL, and PubMed databases from 1985 to 2015. Finally, 14 researches were entered into the study. Psychiatric manifestations may precede the diagnosis of WD and other symptoms related to neurological or hepatic impairment. Early detection of WD is important to prevent catastrophic outcome. Young patients presenting with psychiatric presentations along with abnormal movement disorder, seizure, or conversion-like symptoms should be evaluate for WD even if signs and symptoms are typically suggestive of schizophrenia or manic episode. An interdisciplinary approach with good collaboration of psychiatrists and neurologists is crucial for WD because early diagnosis and management without delay is an important for good prognosis.

Key words: Neuropsychiatric, psychosis, psychosomatic, schizophrenia, Wilson disease

INTRODUCTION

Wilson’s disease (WD) is a neurodegenerative disorder due to copper metabolism.[1] Dr. Wilson first described WD in 12 patients in 1912.[2] The disease is described by excessive accumulation of copper in different tissues and primarily affects the liver and the brain.[3] The lifetime prevalence is at around 1/30,000; however, a recent research of frequency with which the abnormal accumulation of copper is distributed in the body found to be higher.[4] There are neurological, psychiatric, hepatic, or dermatological signs and symptoms. The clinical presentation may vary from the very early period of the disease, which is usually a psychiatric manifestation, to the very late period of the disease, which may show signs of liver or neurological dysfunction. The symptoms of WD usually appear in adolescence or young adulthood, but WD can appear at any age, even in the elderly. The neurological symptoms may be subtle and include tremor, movement disorders, or changes in gait.[5] Psychiatric symptoms can occur in more than 50% of patients with WD at some point in their lives.[6] Psychiatric manifestations may precede the diagnosis of WD by years and may include affective disorders, schizophrenia, and delusional disorders. A case report of a patient who presented with a suicide attempt due to commanding auditory hallucination and was diagnosed with WD after 19 years of presenting symptoms will be described in this paper.
gene related to WD appears points to a possible higher prevalence of 1/7026. Clinically, WD generally appears between the ages of 10 and 20 years. The first presentations of the WD are hepatic (45% of the cases), neurological (35%), or psychiatric (10%). WD is often accompanied by psychiatric manifestations, which may precede signs and symptoms indicating neurological deficit. More than 50% of patients with WD exhibit psychiatric disorders and a large proportion of patients undergo psychiatric management.

The most common psychiatric presentations of WD are mood disorders, disinhibition, behavioral disorders, personality disorders, and cognitive impairment. The prevalence of psychiatric disorders in patients with WD varies widely (major depressive disorder, 4%–47%; psychosis, 1.4%–11.5%). Certain ATP7B gene mutations may correlate with specific personality traits that reflect these disorders. Schizophrenia-like psychosis and delusional disorder are rare forms of psychiatric manifestations of WD, and only a few cases have been reported in the literature. Furthermore, many persons with WD will have psychiatric manifestations develop after diagnosis and initiation of management or after relapses.

When psychiatric manifestations preceded hepatic or neurological involvement, the average time between the psychiatric presentations and the diagnosis of WD was 864.3 days. Knowledge of the psychiatric aspects of WD is essential for psychiatrists and other medical clinicians and allied health professionals practicing in the general medical hospital. The objectives of this article are to review case reports whose subject is the incorrect diagnosis of schizophrenia or schizophrenia-like syndrome in patients with WD and to detail one case of this mismanagement of the disease.

### CASE REPORT

A 35-year-old Iranian woman from a rural background and lower socioeconomic class (education up to the 6th standard) presented to the consulting psychiatrist in an academic general hospital. She was eventually admitted to the Intensive Care Unit after a suicide attempt, in which she ingested alkali-based detergent. The woman was intubated and placed on a ventilator for aspiration pneumonia and bacterial pneumonitis that occurred after the initial chemical pneumonitis. Communication was possible by writing. All four of her limbs exhibited involuntary jerking movements, primarily on the left side. She also complained of infrequently hearing voices that were commanding and persecutory in nature. In this admission, the laboratory results for liver function testing were as follows: total bilirubin = 2.4 mg/dl, direct bilirubin = 1.0 mg/dl, aspartate aminotransferase = 11 IU/L, and alanine aminotransferase = 31 U/L. Thrombocytopenia was present (plt: 44,000, 39,000, 12,000/mm³). Serum ceruloplasmin was markedly reduced at 0.164 g/L (reference values: 0.204–0.407 g/L); copper concentrations were significantly decreased at 50 µg/dl (reference range, 70–153 µg/dl), as 24-h urinary copper was high at 240 µg/24 h (volume: 1690 cc) (reference value up to 80 µg/24 h; WD >100 µg/24 h). Ophthalmological examination demonstrated the initial development of Kayser–Fleischer (KF) rings in the clear anterior segment of the cornea. WD was diagnosed after laboratory tests; furthermore, the patient received a diagnosis of the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition Axis I psychotic disorder due to WD, with delusions. Penicillamine was administered by her main physician, a gastrointestinal specialist. Unfortunately, the patient died after 11 days due to chemical burning and its complications caused by her ingestion of detergent. Review of her history showed during her adolescence, she experienced seizures (at the age of 12 years) and received treatment with sodium valproate; she did not have a developmental retardation in comparison with others her age until the age of 9 years old. At that time, she had some behavioral problems in school and was eventually expelled from school at the age of 12 years. At the age of 17 years, the first set of severe psychiatric signs and symptoms surfaces, including negativism, delusions, and impaired verbal communication. Antipsychotic medications she was prescribed included haloperidol 20 mg/day and nortriptyline 100 mg/day. No significant improvement in her condition occurred; because of this, other antipsychotic therapies (including clozapine) were used over a period of almost 19 years; she was admitted to eleven different psychiatric wards.
during this time, with different diagnoses emerging [Table 1]. In addition, she experienced multiple side effects with different antipsychotics at different times – severe akathisia with haloperidol, tremor with risperidone, and cytopenia due to myelosuppression with clozapine [Table 2].

In 2013, magnetic resonance imaging of the brain showed atrophic changes in the temporal lobe. Since no appropriate improvement was seen with medications, and the patient’s suicidal ideations continued, a course of electroconvulsive treatments was planned and administered during her first, second, fourth, fifth, seventh, ninth, and also tenth admissions to the psychiatric ward. During the patient’s last admission to the psychiatric ward, she experienced tremor in her extremities and hands, right-sided pitting edema in her right limb, falling, and swelling and ecchymosis in her right knee. Nurses said that she had hysterical movements such as pseudoseizure and voluntary falling in the ward (the falling, in retrospect, was probably involuntary rather than voluntary). Numbers from liver function testing were as follows: aspartate aminotransferase = 14 IU/L (reference range, 8–37 IU/L), alanine aminotransferase = 8U/L (reference range, 8–37 IU/L). Other laboratory values included the following: calcium, 8.1 mg/dl (reference range, 8.6–10.3 mg/dl); phosphor, 4.67 mg/dl (reference range, 2.6–4.5 mg/dl). Thrombocytopenia was also present (plt: 40,000/mm³). A hematologist was consulted due to the presence of thrombocytopenia and advised further consultations with a hematologist.

Table 2: Significant psychiatric/physical symptom and significant events

| Time               | Psychiatric/physical symptom and events                  |
|--------------------|--------------------------------------------------------|
| September 23, 2002 | Clozapine-induced cytopenia                             |
| September 23, 2007 | Visual hallucination, auditory hallucination            |
| October 7, 2007    | Clozapine-induced BM suppression clozapine and start of risperidone |
| October 17, 2008   | Head tremor, tremor in trunk, and no good response to questions |
| November 7, 2011   | Tremor in the upper limbs without decreased consciousness and without incontinency |
| January 6, 2013    | History of two episode of pseudoseizure                 |
| January 15, 2013   | Generalized tonic-clonic seizure consultation with neurologist rule out metabolic problem, EEG, brain MRI after stability |
| January 19, 2013   | Decreased level of consciousness and disorientation to TPP, confusion, and delirium |
| April 13, 2012     | Cortical thinning of temporal lobe, temporal atrophy    |
| April 20, 2012     | No response to risperidone, severe akathisia with haloperidol |
| January 20, 2013   | Decreased calcium (7/2) internist neurologist           |
| July 29, 2013      | Thrombocytopenia. Rule out KF rings                      |

EEG = Electroencephalogram; MRI = Magnetic resonance imaging; TPP = Thiamine pyrophosphate; KF = Kayser–Fleischer; BM = Bone marrow

The patient was referred to the general hospital for monitoring of her medical problem. She arrived at the emergency department 9 months later, with the suicide attempt explained at the beginning of this case study.

**DISCUSSION**

The presented case of WD has several specific manifestations that distinguish it from most other patients with WD. Although treated with antipsychotics, she had periods of remission and relapse, and never was symptom-free. Similar cases have been described elsewhere, but to the best of our knowledge, this case represents the longest period over which a psychotic disorder was diagnosed and treated without uncovering the underlying cause of the symptoms (19 years). Such a delay is particularly tragic as favorable outcomes depend on early discovery.[9] The patient died after her suicidal attempt that was spurred by auditory hallucinations. She developed tremors after initiation of injectable haloperidol and severe akathisia with risperidone. She had abnormal involuntary choreoathetoid limb movements and jerky movements that were involuntary and misinterpreted as side effects of antipsychotics but that actually represented the clinical manifestation of WD.

Traditional or atypical antipsychotics have been used with the overall warning that these patients are very sensitive to antipsychotics and prone to develop multiple extrapyramidal side effects.[10] Unfortunately, the neurologist consulted during the first admission adjusted the dose of her anticonvulsant medication (against generally accepted recommendations) due to her previous seizures, as he was unaware of the underlying cause of her condition.

Although psychiatric features are not rare in WD, their association with seizures is rare.[11] Our patient with predominantly temporal atrophy had epileptic seizures and then psychosis. Another case study reported on three patients with psychiatric symptoms who had predominantly white matter lesions in the frontal lobe. The early manifestations were psychiatric symptoms and epileptic seizures with or without secondary generalization. The present observation found that early onset of psychiatric presentation and seizures is common in WD with white matter lesions in frontal lobes.[11] A study from India that evaluated 350 patients with WD reported psychosis (of the schizophreniform type) in three cases only.[12] Other literature have also reported a low prevalence of psychosis – 1 out of 30 cases,[10] 8 out of 70 cases,[11] 3 cases of organic delusional disorder out of 45 patients,[13] 2 out of 195 cases,[14] and 2 out of 42 cases.[8] The prevalence of psychosis in persons with WD varies from 0% to....
Table 3: Case reports of WD with psychosis in psychiatric presentation

| Year | Author | Patient’s age | Psychiatric symptom or diagnosis and initial manifestation | Psychopharmacological management | Time form initial presentation to diagnosis (days/years) |
|------|--------|---------------|----------------------------------------------------------|-----------------------------------|--------------------------------------------------------|
| 1984 | Jayaswali[15] | 18/male | Gradual social withdrawal (e.g., avoiding contact with friends and relations), muttering to self, sleeplessness, and fear of people watching him, and planning to harm him and his family members | Chlorpromazine, Phenobarbitone, Carbamazepine, Trihexoperazine | 4 years |
| 1985 | Modai[30] | 22/female | Schizophrenia | Penicillamine 750 mg, Vitamin B6 50 mg, Haloperidol 3 mg, Trihexyphenidyl 6 mg daily | 4015 days |
| 1995 | Chin-Chang Huang[11] | 20/female | Nervousness, irritability, ideas of reference and persecution schizophrenia diagnosis in another hospital, L hand tremor, L hemiparesis, L limb dystonia, seizures | D-penicillamine daily dose of 1500 mg, Phenytoin, carbamazepine, and haloperidol | 3 years |
| 1995 | Chin-Chang Huang | 26/male | L hand dystonia, seizures, idea of persecution, auditory Hallucination, bizarre behaviors Scanning dysarthria, drooling Rigidity, and increased tendon Reflexes | Electroconvulsive therapy, D-penicillamine 750 mg daily, Intravenous phenytoin, Carbamazepine 500 mg, Phenytoin 300 mg, Vitamin B6 50 mg daily | 12 years |
| 2003 | Sagawa[44] | 23/female | Nervousness, irritability, compulsive acts, paranoid delusion Tremor, gait disturbance, dystonia, seizures, catatonia, akinetic-rigid syndrome Impression of schizophrenia | Pharmacological treatment, D-penicillamine | 7 years |
| 2006 | Hubert Michał[44] | 32/male | Loss of balance, postural, and kinetic tremor in the upper limbs and occasional diplopia substantial cognitive impairment and poor insight A complex and coherent delusional system. He refused any treatment in the belief that his family wanted to kill him | Penicillamine 500 mg/day, Zinc 800 mg/day and propranolol 30 mg/day, Olanzapine (15 mg/day), Perazine (150 mg/day) | 2 years |
| 2006 | Jukić and Titlić[26] | 26/male | Acute psychosis Abnormal involuntary Choreaathetoid limb movements | Haloperidol | Few days after initiation of neuroleptic therapy |
| 2006 | Wichowicz[46] | 31/male | Irritable, psychosis, aggressive | Olanzapine, piperazine | 730 days |
| 2006 | Chahine[45] | 21/female | Asymptomatic, KF rings, genetic testing Personality change, irritability, psychosis, and suicide attempts Asymptomatic, KF rings, and genetic testing | Ziprasidone, fluoxetine, trientine | 2555 days |
| 2008 | Spyridi[27] | 34/male | Loss of balance, postural and kinetic tremor in the upper limbs, and occasional diplopia Persecutory delusion | Risperidone, Chlordiazepoxide | 1095 |
| 2011 | Somsahbra Chattopadhyay[49] | 20/male | Sudden jerky, involuntary movement of head, trunk, and all four limbs for the past 6 months and abnormal behavior for the past 1 month Hearing voices which were commanding and persecutory in nature Visual hallucination, somatic passivity, and somatic hallucination Thought broadcasting and feature like syndrome of Fregoli | Antiepileptics 300 mg/day zinc sulfate and 10 mg/day olanzapine | 6 months |
| 2012 | Reza Bidaki[25] | 33/male | Psychotic disorder | Mood stabilizer (carbamazepine, lithium), TCA (amitriptyline), benzodiazepines (chlordiazepoxide, clonazepam), piracetam, and trihexyphenidyl | Almost 10 years |

Contd...
11.5%. Since the early presentations of WD, few case reports have documented the occurrence of psychosis in patients with WD. However, over the last two decades, case reports/series have proliferated describing psychosis in patients with WD.

In our case, WD was determined by the following positive findings: (I) Presence of KF ring (II) high urinary copper level, and (III) low serum ceruloplasmin level. The gold standard for WD diagnosis is by liver biopsy with quantitative copper evaluation (copper values >3.1 μmol/g of dry liver weight). Liver biopsy was not performed for several reasons: the invasiveness of the procedure, the inability to estimate copper in dry liver tissue with the equipment available at our center, and the fact that approval for biopsy was not given by the patient’s brother (her caretaker). The combination of neurological symptoms, KF rings, and a low ceruloplasmin level is considered sufficient and appropriate for the diagnosis of WD.

Copper is considered essential for brain function and its importance in various psychiatric illnesses has been explored. Initial researches showed increased levels of copper in the hair and plasma of patients with schizophrenia compared to control groups. Abnormalities of smooth pursuit eye movements, long time considered a mark in schizophrenic patients, have been described in WD. Some researches found that an increased copper level was related to the use of psychotropic drugs, such as long-acting antipsychotic use in schizophrenic patients or anticonvulsant treatment in women with epilepsy. The copper hypothesis of schizophrenic patients was abandoned in 1980 without ever being totally refuted. Wolf found that patients with schizophrenia had serum copper levels that were increased by 24% and serum ceruloplasmin levels elevated by 20% compared to control group. It was postulated that an excess of copper may affect dopamine activity through multiple copper-dependent enzymes. Another recent literature suggested that dopamine gene polymorphism may be playing a significant role in the neuropsychiatric manifestations in WD.

Co-occurrence of schizophrenic-like symptoms with WD is rare, and there are just a few case reports of WD with this type of psychiatric presentation. Table 3 provides an overview of the case reports about this issue.

### Table 3: Contd..

| Year | Author                  | Patient’s age | Psychiatric symptom or diagnosis and initial manifestation                                                                 | Psychopharmacological management                                                                 | Time from initial presentation to diagnosis (days/years) |
|------|-------------------------|---------------|-------------------------------------------------------------------------------------------------|---------------------------------------------------------------------------------------------------|--------------------------------------------------------|
| 2014 | Sandeep Grover          | 12/male       | Abdominal distension associated with dull aching continuous pain for 1 month                   | Penicillamine risperdone 1 mg/day                                                               | 1 month                                                |
| 2014 | Dragan Krstic           | 22/male       | Social withdrawal, reduction of verbal communication, increased hostility toward family members  | Anxiolytics and psychotherapy Risperdone and clozapine Paroxetine, mirtazapine, bupropion         | 2 years                                                |

KF – Kayser–Fleischer; TCA – Tricyclic antidepressant

**CONCLUSIONS**

Patients with psychiatric symptoms with clues such as history of jaundice, family history of neuropsychiatric disorders, and sensitivity to typical/atypical antipsychotics should be evaluated for WD to avoid delay in diagnosis and associated morbidity. Although such patients are more commonly seen in neurological and hepatological clinics, psychiatrists must keep in mind a high level of suspicion, and consider WD in instances where the first presentation of illness is psychiatric in nature.

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**Conflicts of interest**

There are no conflicts of interest.

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Wilson's disease as schizophrenia

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