Wilson Disease Presenting with New Clinical and Biochemical Lab Findings: A Case Report and Review of Literature

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Abstract

Introduction: Wilson disease, an inherited autosomal recessive disorder of copper metabolism, is characterized by pathological copper accumulation. The disease is potentially treatable and usually caused by mutations in the ATP7B gene, encoding the transmembrane copper-transporting adenosine triphosphatase. Despite its well-documented neurological and hepatic manifestations, electrolytes disturbances, including hypermagnesemia, remained under-reported and rarely associated with the disease.

Case Presentation: A married Asian, Pakistani Punjabi, male from a middle-class family in his late 20s was admitted to the neurology ward of a tertiary care hospital with no previous medical history of disease. Initial assessments illustrated vital signs: body temperature 98°F, blood pressure 120/70 mmHg, random blood glucose level 81 mg/dl, pulse 82 beats per minute, oxygen saturation 98%, and hydration status (input: 1500 ml, output: 1000 ml). Physical examination revealed pallor on face and normal eyes, but mild silvery-brown edges in the corneas of both eyes (Kayser-Fleischer rings). Screening with mini nutrition assessment - short form, indicated malnourishment with a score of 4 (score: 0–7 signifies malnourished), accompanied by a body mass index of 20.43 kg/m². The subjective global assessment form showed a 10.93% weight loss within one month. Hypermagnesemia was prominent, with serum magnesium levels exceeding 2.2 mg/dl. Liver function tests revealed elevated aspartate transaminase levels.

Conclusion: Wilson's disease patients could develop severe malnutrition after the onset of the disease, hypermagnesemia, a dry white tongue, mouth sores despite good oral hygiene, and fatigue due to low oxygen supply, as indicated by low levels of haemoglobin in the blood.

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Introduction

Wilson disease (WD) is an inherited disorder of copper metabolism, characterized by copper accumulation. It is an autosomal recessive disorder caused by mutations in genes [1]. The disease is potentially treatable and usually results from heterozygous or homozygous mutations in the ATP7B gene, which encodes the transmembrane copper-transporting adenosine triphosphatase (ATPase) [2 – 4]. This gene also regulates copper excretion into bile and its delivery for the functional synthesis of ceruloplasmin (CP), a copper-containing protein. Mutations in this gene lead to excessive accumulation of copper in the brain, liver, and other organs. The severity of WD can vary during its clinical course, but progressive liver disease is common.

Patients typically present with psychiatric and neurological symptoms. Symptoms of WD usually occur between the ages of 5 and 35 years. Although WD is rare, with an estimated prevalence of 1 in approximately 30,000 individuals [3], it can manifest in individuals with certain genetic disorders and is treatable if diagnosed early and managed properly [2].

There are several difficulties in diagnosing WD due to delays in reporting the initial symptoms and establishing a diagnosis. Symptoms are usually straightforward in children, often presenting as liver manifestations. In adolescents, symptoms may include disturbances in movement and personality, along with the classical biological triad of low CP levels and elevated urinary copper excretion over 24 hours. Diagnosing such rare diseases remains challenging and can often take more than two years [5]. Although the disease has a genetic basis, predicting the relationship between genotype and phenotype continues to be difficult. Diagnosis of WD relies on a combination of biochemical, histological, clinical, and genetic findings. However, the current available procedures and diagnostic tests often fail to detect the disease due to their limited specificity and sensitivity [6]. Biochemical findings, such as serum CP concentrations and urinary copper excretion, may also overlap with levels found in healthy individuals [7].

This case report presents a rare case of WD that was initially mistaken for muscular dysfunction. The patient exhibited severe malnourishment, hypermagnesemia, dry-white tongue, and mouth sores, yet no kidney dysfunctions. This case report aimed at highlighting these novel features to broaden the spectrum of WD for early diagnosis and management. The format of the case report has been checked and validated in accordance with the published guidelines [8].

Case Presentation

A married Asian, Pakistani Punjabi, male from a middle-class family with a low level of education, living in a poor housing area in his late 20s, was admitted to the neurology ward of a tertiary care hospital. He had no previous medical history relevant to any non-communicable or communicable diseases. No one in his family had a history of Wilson's disease, hypertension, chronic kidney disease (CKD), or cardiovascular diseases (CVDs), although one family member was diagnosed with type 2 diabetes. He had not taken any medication before the onset of his symptoms. His reported presenting complaints included lack of mobility, dysphagia, and seizures.

The initial assessments showed his vital signs: body temperature (98°F), blood pressure (120/70 mmHg), random blood glucose level (81 mg/dl), pulse (82 bpm), oxygen saturation (98%), and hydration status (input: 1500 ml, output: 1000 ml). Physical examination revealed paleness on his face and normal-looking eyes with no pallor or dullness, but thin silvery-brown edges- Kayser-Fleischer rings- were seen in both corneas under magnification. The initial diagnosis was muscular dysfunction caused by sudden shock from stress. The provisional diagnosis suspected WD due to dystonia and the slight silvery appearance around the corneas. The treatment involved oral drugs including baclofen, trihexiphenidyl, clonazepam, carbamazepine, metformin, xenazine, and escitalopram.

Screening with the Mini Nutritional Assessment short form (MNA-SF) indicated malnourishment with a score of "4" (0–7 signifying malnourished, 8–11 at risk of malnourishment, 12–14 normal nutritional status), despite a body mass index (BMI) of 20.43 kg/m^2. The Subjective Global Assessment (SGA) form showed a 10.93% weight loss within one month during the stay at the tertiary care hospital, suggesting severe malnourishment. Biochemical assessments revealed no signs of hypercholesterolaemia, hypertriglyceridaemia, hypernatraemia, or hyperkalaemia. Cholesterol levels were < 250 mg/dl, and triglycerides were < 150 mg/dl, both within normal ranges. Complete blood count (CBC) results showed no abnormalities in white blood cells WBCs, RBCs, haematocrit, or platelets. Although haemoglobin and red cell distribution width (RDW) levels were low, red cell distribution width-coefficient of variation (RDWc) was high. Serum electrolyte tests identified hypermagnesaemia (serum magnesium 2.52 mg/dl), with magnesium levels exceeding the recommended range, while sodium and potassium levels remained normal. Renal function tests (RFTs) demonstrated normal creatinine and urea values. However, liver function tests (LFTs) indicated elevated aspartate aminotransferases (AST), while alanine aminotransferases (ALT), bilirubin, albumin, and prealbumin remained within normal ranges. Serological testing was negative for hepatitis B surface antibody (HBsAb) and hepatitis B core antibody (HBcAb), ruling out hepatitis B. Clinical examinations revealed pale skin, good oral hygiene, and a dry white tongue; lips, nails, and hair appeared normal. Gastrointestinal function testing showed appetite suppression, nausea, altered bowel functions, and distorted taste and smell, but no acidity or indigestion. Additional symptoms included mouth sores, early satiety, dystonia, fatigue, weakness, dysphagia for solid foods, and seizures. The patient showed no signs of depression or anxiety, remaining cheerful during his hospital stay. Dietary assessment revealed excessive carbohydrate intake (> 220 g/day) and moderate fat intake (> 65 g/day), while protein intake was markedly low (< 22 g/day), with excessive consumption of whole fruits and branded fruit drinks.

The findings aligned with the suspected diagnosis of WD's, as the symptoms matched its clinical features. The treatment plan was focused on reducing intake of copper-rich foods to prevent copper overload until the condition was managed. The usage of zinc supplements was encouraged to inhibit intestinal copper absorption. Additionally, avoiding caffeine, which might worsen symptoms, and using copper chelating agents like penicillamine to lower copper levels in the body were recommended. The patient was admitted for two months and showed a somewhat slower response to treatment because he did not adhere to the dietary guidelines proposed by the dietitian and lost more than 10% of his body weight during this period.

Discussion

As WD is an inherited disorder caused by a recessive gene [1], in this case, the patient had no family history of diseases such as WD, CKD, CVD, and hypertension. However, a close family member had a history of type 2 diabetes. Serum copper concentrations are not usually measured in clinical practice, and probably no biomarkers can reliably assess copper status. Although copper levels might be affected by factors such as inflammation, infection, pregnancy, oestrogen levels, and various cancers. The normal serum concentration of copper was estimated to be 10 to 25 µmol/l or 63.5 to 158.9 µg/L. Meanwhile, normal CP levels were typically between 180 and 400 mg/L [9].

Malnutrition was not a common feature in adults suffering

from WD, but this patient experienced severe weight loss leading to under-nutrition. Nevertheless, it was rarely observed in children with WD [10]. WD was typically associated with prominent psychiatric symptoms and an increased risk of sleep disorders and cardiac issues. Copper deposition might also cause mental retardation, neuropathy, keratoderma, enteropathy, deafness, ichthyosis, and congenital disorders of glycosylation [11]. Approximately 20–69% of WD cases presented with dystonia, 85–90% with Kayser-Fleischer rings, 55–72% with postural tremors, and 4–15% with seizures, which were more common in younger patients [12].

Table 1: Insights of unique findings of the current case and evidence from literature

	Unique case findings	Literature findings
Malnutrition	Severe malnutrition observed in the patient	Malnutrition observed in the children with WD [10]
Hypermagnesemia	Hypermagnesemia observed in patient's findings with normal sodium, potassium and chloride levels	Persistent hypokalemia, hypomagensemia and hyperchloremia reported in the patients [19]
Dry white tongue	Dry-white tongue was observed during clinical examination	No literature findings were available on the symptom of dry-white tongue in WD clinical presentation
Kidneys	No kidney dysfunction was noticed during the clinical and biochemical examinations	Renal degeneration of the tubules might result due to multiple-organ dysfunction [26, 27]
Mouth sores	Mouth sores were prominent in the patient but oral hygiene was good without any periodontal disease although he was not taking D-penicillamine	People of WD might develop oral complications due to the usage of D-penicillamine [28]
LFTs	In current findings, only AST levels were elevated with normal ALT, bilirubin, prealbumin and albumin levels	Acute liver failure is associated in WD [25], patients usually have persistent high levels of AST and ALT with asymptomatic hepatomegaly [13]

Table 1 of this study provided insights on the current and published literature findings related to WD, where numerous new findings showed significant deviations from the previously reported results. Additionally, some findings from this study aligned with the previous findings on WD. Prominent severe malnutrition was observable in the study, as shown in (Table 1), supported by the earlier research on WD [10]. Peculiar findings of hypermagnesemia with normal serum sodium, potassium, and chloride levels have been demonstrated in (Table 1) that refuting the occurrence of persistent hypokalemia, hypomagnesemia, and hyperchloremia [19]. Additionally, the findings of dry tongue in WD have been mentioned in (Table 1), but lacked the supporting literature evidence. Surprisingly, no kidney dysfunction was noticed as demonstrated in (Table 1), although earlier studies reported renal degradation in WD [26, 27]. Furthermore, the findings of mouth sores and deranged levels of LFT biomarkers with supporting evidence [13, 25, 28] were mentioned in (Table 1).

The clinical patterns of WD also demonstrated hepatic symptoms such as persistently elevated serum AST and ALT levels, acute liver failure, fatty liver, acute liver injury, cirrhosis, asymptomatic hepatomegaly, and isolated splenomegaly. Affected individuals might also present with bipolar disorder, depression, personality changes, neurotic behaviors, and psychosis. Additionally, dysrhythmia, cardiomyopathy, hypoparathyroidism, renal abnormalities, including nephrolithiasis, pancreatitis, infertility, and premature osteoporosis, may occur during the disease progression [13]. Copper imbalance was also associated with chronic liver disease that resulted from liver injuries or possibly due to viral hepatitis infection [14].

Although there was no diagnosis of liver failure, liver injury, or cirrhosis for the current patient, only AST levels were higher than their normal values. Negative results of HBsAb and HBcAb in serological testing also eliminated the chances of viral hepatitis B. Liver-related symptoms including weakness, vomiting, itchiness, ascites, and swelling in the legs may also be present in WD [24]. Although this patient did not have any of these symptoms except weakness and fatigue, loss of appetite, nausea, and vomiting were also attributed to the consumption of zinc supplements [15]. Even so, nausea and loss of appetite were the only symptoms presented in the patient.

For estimating WD prevalence, screening for ceruloplasmin can be conducted either in urine, dried blood spots, or by detecting Kayser-Fleischer rings in the eyes. However, these screening methods make it difficult to determine an exact prevalence, as they may overestimate the disease [1]. Taste and smell disturbances were observed in this case, supported by a previously published study [16]. Low levels of haemoglobin and RDWs were notable, although RDWc levels were higher than normal values. As shown in previous research, the odds of haemolytic anaemia (HA) increase when a patient with WD presents with cirrhosis [17].

This case presentation did not show any deviations in potassium and sodium levels. Occurrence of WD with hypokalaemia and muscle paralysis may also occur due to involvement of the distal tubule and acidosis in proximal tubules [18]. Hyperammonemia might also be present in individuals with the disease, possibly due to hepatocellular injury and coagulopathy, yet blood ammonia levels were not investigated for the patient. Patients may also present with persistent hypomagnesaemia, hypokalaemia, low bicarbonates, and hyperchloremia even in the absence of metabolic acidosis [19]. However, instead of hypomagnesaemia, the patient developed hypermagnesaemia, serum magnesium levels > 2.2 mg/dl upper reference value [20], without any indication of acidosis or other associated conditions. The patient had normal levels of serum potassium, bicarbonates, sodium, and chloride in this case report.

Dysphagia was also a neurological manifestation of WD and might persist alongside general weakness, which was also evident in the current case report. The swallowing process involved complex interactions between brainstem nuclei—both motor and sensory—and the cerebral cortex. Lesions affecting these neurological structures could also lead to neurological dysphagia [21]. The patient's reports indicated normal levels of cholesterol and triglycerides, with no evidence of hypercholesterolemia or hypertriglyceridemia. Similar findings had been reported in previous literature,

where cholesterol levels remained within recommended ranges [22]. Oral hygiene was typically a concern in WD patients, as they often had poor oral hygiene, petechiae in the oral mucosa, carious teeth, periodontal disease, and missing teeth [23]. However, the current case did not exhibit such symptoms. Instead, it showed good oral hygiene but the presence of mouth sores. The presentation of a dry, white tongue was a unique finding not consistent with previously published literature.

Conclusion

In conclusion, WD patients can develop severe malnutrition after disease onset, hypermagnesemia, a dry white tongue, mouth sores even with good oral hygiene, and fatigue due to low oxygen supply, as indicated by low haemoglobin levels in the blood. Classical Kayser-Fleischer rings might not be prominent yet; however, these could be thin and noticed with a magnifying glass. Although liver injury or hepatitis were not necessary in lab findings, merely elevated AST might be observed. Dysphagia, nausea, and distorted taste and smell could present in the patients. However, further investigations are needed to establish the causal relationship and natural history of the disease.

List of Abbreviations

Abbreviation	Full Form
ALT	Alanine Aminotransferase
AST	Aspartate Aminotransferase
ATP7B	ATPase Copper Transporting Beta Polypeptide (gene)
BMI	Body Mass Index
CBC	Complete Blood Count
CKD	Chronic Kidney Disease
CP	Ceruloplasmin
CVD	Cardiovascular Disease
HBcAb	Hepatitis B Core Antibody
HBsAb	Hepatitis B Surface Antibody
LFTs	Liver Function Tests
MNA-SF	Mini Nutritional Assessment–Short Form
RBCs	Red Blood Cells
RDW	Red Cell Distribution Width
RDWc	Red Cell Distribution Width–Coefficient of Variation
RFTs	Renal Function Tests
SGA	Subjective Global Assessment
WD	Wilson Disease
WBCs	White Blood Cells

Declarations

Ethical consideration: The patient was willing to participate, as case presentation was the part of corresponding author's hospital visits. At both stages, before taking patient's history and writing this case report, a prior verbal informed consent was obtained from the patient to follow ethical guidelines. Patient's name, phone number, pictures and any identifiable information are not included at any part of the manuscript. The patient willingly provided the informed consent, which was further signed by the corresponding author due to patient's restricted voluntary body movements due to dystonia.

Consent for publication: Written informed consent was obtained from the patient for publication of this case report and any accompanying images in accordance with the Declaration of Helsinki.

Availability of data and materials: The data will be made available by hiding patient's identifying information on justifiable request by the journal to ensure ethical integrity.

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