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Three More Cases of Adult Wilson's Liver Disease from Bangladesh and Review of Literature

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Abstract

Wilson's Disease is an autosomal recessive genetic disease that was first reported more than a century ago. Our knowledge about the disease has since flourished and we now know the in and outs of this infrequent, but not rare disease very well, including treatment of both forms of the disease affecting the liver and the brain. This disease results from accumulation of excess copper in liver and/or brain. Diagnosis may be difficult due to complex disease manifestations. 24-hour urinary copper estimation remains the mainstay of diagnosing Wilson's disease. If untreated, the prognosis is poor. However, there are excellent treatment options for Wilson's disease these days including liver transplantation. Strong clinical suspicion and extensive investigation are important for diagnosis of Wilson's disease. It is therefore important to share experience about individual cases of Wilson's disease in scientific literature to keep peers continuously updated about the varied presentation of this life-threatening condition. Here we report 3 recent cases of adult Wilson's liver disease as well as review of published literature from Bangladesh.

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1. Introduction

Wilson's Disease (WD) or hepatolenticular degeneration is an autosomal recessive disorder of copper transport characterized by accumulation of copper in liver, brain and cornea [1]. WD was first described in 1912 by a Neurology resident Dr. Wilson and the disease bears its name after him [2]. Later in 1948, Dr. J. N. Cumings first elucidated the link between copper and WD [2]. The disease results from mutation of ATP7B gene on chromosome 17, which encodes copper transport P-type ATPase (ATP7B) in the trans-Golgi tissue of hepatocytes [3]. ATP7B transfers intracellular copper to bile and regulates ceruloplasmin synthesis [4]. Incidence of WD is 1 in 30,000 and it can affect people of any age, particularly those between 5 to 35 years of age [5].

2. Pathogenesis

Copper is an essential component of many human proteins [6]. Copper content of adult human body is approximately 100 mg. Against a daily copper requirement of approximately 0.9 mg, humans on an average consume 2-5 mg copper per day [7]. Biliary excretion is the primary route of elimination of this excess copper from the body, where the role of the liver is critical, as it is the principal site of copper homeostasis regulating copper storage and excretion [8, 9]. The amount of copper excreted from the body equals the hepatic copper pool. In WD, copper homeostasis fails leading to accumulation of excess copper in different organs like, liver, brain and cornea.

3. Clinical Features

Hepatic involvement in WD varies widely. Patients usually present with liver cirrhosis (LC). However, patients may also present without features of chronic liver diseases and on the other extreme, may even have signs of fulminant hepatic failure at the time of presentation. Patient's may have Kayser-Fleischer (KF) rings in eyes resulting from accumulation of copper in the Descemet's membrane of cornea [10]. This ring is present in 30-50% patients with pre-symptomatic or hepatic WD and it's absence does not exclude the condition.

4. Diagnosis

Diagnosis of WD remains challenging. Presence of KF ring and low serum ceruloplasmin (<100 mg/L) confirms the diagnosis [11]. However, KF rings may be overlooked in black corneas. Besides, serum ceruloplasmin may be low in advanced liver diseases of variable etiology [12]. Laboratory findings that support the diagnosis of WD include low serum copper, elevated liver transaminases, aminoaciduria and hemolytic anemia [13]. Serum total copper is usually low in WD due to decreased ceruloplasmin in blood [8].

24-hour urinary copper excretion exceeding 100 gm suggests WD, in the absence of cholestatic liver disease. However, challenges of measuring 24-hour urinary are manyfold and include insufficient urine collection, copper contamination of collection device, co-existent chronic kidney disease and overlapping findings with other liver diseases e.g. autoimmune hepatitis, chronic liver disease, acute liver failure, cholestasis etc. [7].

Excess copper in the liver confirms WD, but it cannot be measured without obtaining liver tissue at liver biopsy. The Leipzig scoring system (TABLE 1) [14] that utilizes biochemical, clinical and genetic data and endorsed by European Association for the Study of the Liver (EASL), is used to diagnose WD [5].

TABLE 1. **Leipzig scoring system for diagnosis of Wilson's disease [14].**

Kayser-Fleischer rings		**Liver copper (in absence of cholestasis)**	
Present	2	>250 µg/g	2
Absent	0	50 - 250 µg/g	1
Neurologic symptoms (or typical brain MRI)		Normal (<50 µg/g)	-1
Present	2	Rhodamine-positive hepatocyte on biopsy*	1
Absent	0	Urinary copper (in absence of acute hepatitis)	
Serum ceruloplasmin		Normal	0
Normal (>0.2 g/L)	0	1.2x ULN	1
0.1 - 0.2 g/L	1	>2x ULN	2
<0.1 g/L	2	Normal, but >5x ULN after D-penicillamine	2
Coombs-negative hemolytic anemia		Mutation analysis	
Present	1	Two chromosome mutations	4
Absent	0	One chromosome mutations	1
		No mutation detected	0
Total score		Evaluation	
≥ 4		Diagnosis highly likely	
2.3		Diagnosis probable, more tests needed	
≤ 1		Diagnosis very unlikely	

* If no quantitative liver copper available

5. Treatment

WD patients with Nazer prognostic score (TABLE 2) [15] <7 are treated medically, while score >9 is indication for liver transplantation. Those having score between 7-9 remain in the gray zone and must be assessed individually as to whether they are more likely to benefit from medical intervention or liver transplantation [16].

TABLE 2. **Nazer prognostic score for Wilson's Disease [15].**

Laboratory Measurement	**Score (in points)**					
	Normal Value	**0**	**1**	**2**	**3**	**4**
Serum bilirubin	0.2 - 1.2 mg/dL	<5.8	5.8 - 8.8	8.8 - 11.7	11.7 - 17.5	>17.5
Serum asparatate transferase (AST)	10 - 35 IU/L	<100	100 - 150	151 - 200	201 - 300	>300

Prolongation of prothombin time (seconds)	-	<4	4 - 8	9 - 12	13 - 20	>20

Dietary copper restriction is an important part of WD treatment. Diet rich in copper e.g., chocolate, shellfish, nuts, liver and mushroom and copper cookware must be avoided [17].

Copper chelators e.g., D-penicillamine and trientine excrete the excess copper by binding to copper in blood and tissues directly. However, they can cause bone marrow suppression and proteneuria and therefore treatment with copper chelators should be monitored closely. Between D-penicillamine and trientine, the latter is preferred due to similar efficacy, but better safety profile [18]. Zinc is also used alone or in combination. It inhibits copper absorption from the intestine and is safe, causing nausea and epigastric pain in 10% patients [19]. Zinc can be used in pre-symptomatic patients too. Pyridoxine is added to D-penicillamine as D-penicillamine causes pyridoxine deficiency thus worsening neurological manifestations of WD.

6. Prognosis

If left untreated WD can be fatal which results from complications of liver and/or neurological disease. Nazer prognostic score is used to determine prognosis of WD [16].

7. Reports of 3 Cases of Adult Wilson's Liver Disease

Our first patient was a 45-year-old, middle-aged male who presented with vague complaints like right upper abdominal quadrant discomfort and dyspepsia. He was a known diabetic for 5 years. When he evaluated his serum bilirubin was 0.71 mg/dL, serum alanine aminotransferase (ALT) 105 U/L, serum albumin 4.5 g/dL, INR 1.08, fasting blood sugar 7.5 mmol/L and HbA1c 7.4%. He tested positive for anti HBc (Total), but negative for HBsAg, anti-HCV, HBV DNA, ANA, anti-LKM-1, ASMA and AMA. His abdominal ultrasonography and endoscopy of upper gastro-intestinal tract (UGIT) were both normal. Fibroscan was done where controlled attenuation parameter (CAP) or hepatic steatosis was 296 dB/m and liver stiffness measurement (LSM) or hepatic fibrosis was 9.7 kPa. His serum ceruloplasmin was 25 mg/L, 24-hour urinary copper 143.8 µg/day and IgG 19.4 gm/L. There was no KF ring on Slit Lamp ophthalmic examination. Since his hepatic fibrosis was significant and the diagnosis of WD was not clear cut, we decided to do a liver biopsy.

A 1.4 cm × 0.2 cm linear tan-brown, linear liver tissue was obtained at percutaneous, abdominal ultrasonography guided liver biopsy done with Tru-Cut biopsy needle (Becton Dickenson and Co., NJ, USA). On microscopic examination, there was moderate distortion of lobular architecture with moderate dysplasia in hepatocytes and moderate steatosis. Mononuclear infiltration of portal tracts mostly with lymphocytes and some plasma cells along with lymphoid aggregates were present. Focal area showed mononuclear infiltrate of the hepatic lobules. The hepatocytes had glycogenated nuclei, inflammation and variable hepatocellular anisonucleosis. Clusters of hepatocytes with granular eosinophilic cytoplasm resulting from increased number of mitochondria were also seen. These findings were consistent with WD.

Based on moderate piecemeal necrosis (<50% portal tracts) i.e. score 3, confluent necrosis in some areas *i.e.* score 2, focal necrosis (3-4/10 HPF) *i.e.* score 2 and moderate portal inflammation in all portal areas *i.e.* score 3, histological activity index (HAI) score was determined 10/18 and with fibrous expansion of most portal areas without short fibrous septa, fibrosis score was determined 2.

Besides, based on steatosis (approx. 35% hepatocytes) i.e. score 2, lobular inflammation (6 foci/200x) i.e. score 3 and ballooning of many hepatocytes i.e. score 2, NAFLD activity score (NAS) was determined 7/8 and due to perisinusoidal and portal fibrosis, fibrosis score was calculated to be 2 (FIG. 1). Our final diagnosis was WD with metabolism dysfunction-associated steatotic liver disease (MASLD) with occult hepatitis B virus infection.

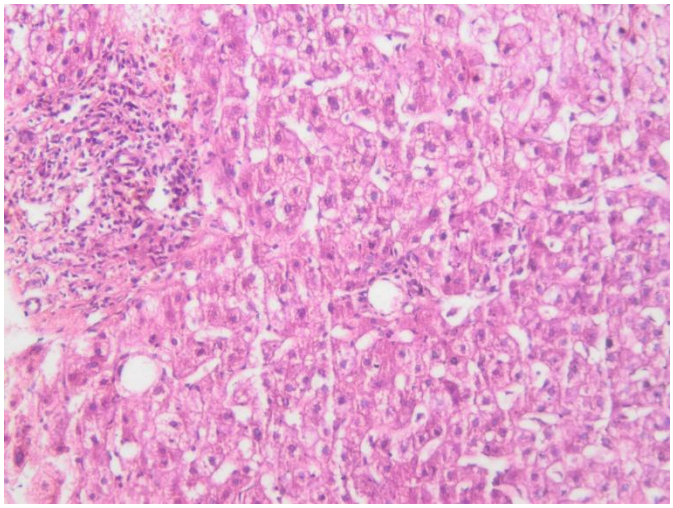

FIG. 1. **H&E 100x, B. H&E 200x: Moderate distortion of lobular architecture with moderate dysplasia in hepatocytes with moderate steatosis. Mononuclear infiltration of portal tracts mostly with lymphocytes and some plasma cells along with lymphoid aggregates are present. Focal areas show mononuclear infiltrate of the hepatic lobules. The hepatocytes have glycogenated nuclei, inflammation and variable hepatocellular anisonucleosis. Clusters of hepatocytes with granular eosinophilic cytoplasm resulting from increased number of mitochondria are also seen. There is moderate piecemeal necrosis (<50% portal tracts), confluent necrosis in some areas, focal necrosis (3-4/10 HPF), moderate portal inflammation in all portal areas and fibrous expansion of most portal areas without short fibrous septa. There is also steatosis (approx. 35% hepatocytes), lobular inflammation (6 foci/200x), ballooning of many hepatocytes as well as perisinusoidal and portal fibrosis.**

The patient was put on D-penicillamine, zinc acetate, pyridoxin and resmetirom along with lifestyle modification and insulin for management of diabetes mellitus (DM). He was also strongly advised to abstain from diet rich in copper.

Our second patient was a middle-aged lady, in her mid-fifties whose incidental finding on abdominal ultrasonography was grade II fatty liver. She was a known diabetic for many years which was controlled on dietary measures and insulin. Her Serum bilirubin was 0.3 mg/dL, ALT 242 U/L, serum albumin 35.8 g/L, INR 1.21 and HbA1c 6.1%. Fibroscan revealed CAP 300 dB/m and LSM 7.9 kPa. She tested negative for HBsAg, anti-HBc (Total), anti-HCV, ANA, ASMA, anti-LKM-1 and AMA. We advised her lifestyle modification.

However, her ALT remained persistently elevated more than 2-3 times above the upper normal limit on repeated follow-ups; although her diabetes was well controlled. At this point, we added obeticholic acid orally, daily but to no yield. After 6 months of follow-up with the patient on pharmacologic plus lifestyle interventions, but no normalization of ALT, we decided to evaluate her further. Her follow-up fibroscan revealed a 'mixed picture' with CAP improving from baseline to 260 dB/m, but LSM progressing to 9.1 kPa. Further investigations revealed serum ceruloplasmin 170 mg/L, 24-hour urinary copper 110 µg/day and presence of KF rings in both eyes. We planned to do liver biopsy, but the patient refused. We added D-penicillamine and zinc to her drug list and expanded her dietary restrictions to exclude copper containing foods.

Our third patient is also a middle-aged female, also in her mid-fifties, who was refereed to us as a case of cryptogenic liver cirrhosis. With her, our main challenge was to identify the etiology of her liver condition. Her serum bilirubin was 0.4 mg/dL, ALT 98.3 U/L, serum aspartate aminotransferase (AST) 128.9 U/L, INR 1.31, serum albumin 30 g/L and AFP 4.95 IU/ml. Abdominal ultrasonography revealed coarse hepatic parenchyma with splenomegaly. She had erosive antral gastritis at endoscopy of UGIT. Fibroscan confirmed the diagnosis of liver cirrhosis with LSM 16.9 kPa and CAP being 216 dB/m. It was also therefore confirmed at fibroscan that the underlying cause was not MASLD. She was negative for HBsAg, anti-HCV, ANA, ASMA, anti-LKM-1 and AMA. However, her anti-HBc (Total) test was positive, but HBV DNA was negative. Her serum ceruloplasmin was 110 mg/L and 24-hour urinary copper 228.5 µg/day. There was no KF ring on Slit Lamp ophthalmic examination.

We decided to do a liver biopsy, which was done with a Tru-Cut biopsy needle (Becton Dickenson and Co., NJ, USA) percutaneously under abdominal ultrasonography guidance. A 1.4 cm × 0.2 cm linear tan-brown, linear liver tissue was obtained. On microscopic examination, mononuclear infiltration of portal tracts mostly with lymphocytes and some plasma cells along with lymphoid aggregates were present. Focal area showed mononuclear infiltrate of the hepatic lobules. The hepatocytes had glycogenated nuclei, inflammation and variable hepatocellular anisonucleosis. Mallory-Denk bodies (MDB) were seen occasionally. Clusters of hepatocytes with granular eosinophilic cytoplasm resulting from increased number of mitochondrea were also seen. Copper and hemosiderin pigment deposition within periportal hepatocytes and Kupffer cells were seen (FIG. 2). The diagnosis of WD was thus confirmed.

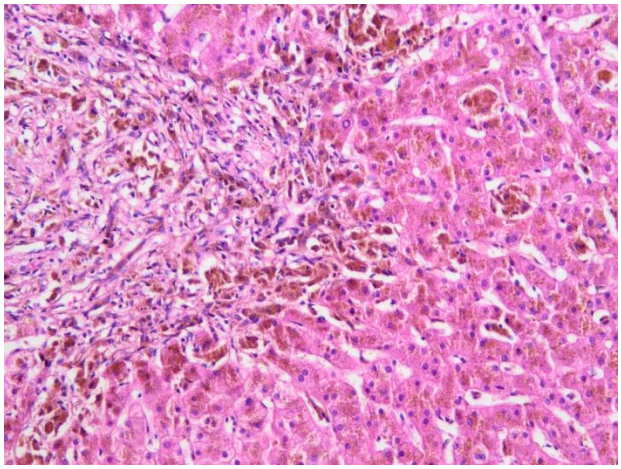

FIGURE 2. **H&E 100x, B. H&E 200 x: Mononuclear infiltration of portal tracts mostly with lymphocytes and some plasma cells along with lymphoid aggregates are present. Focal areas show mononuclear infiltrate of the hepatic**

lobules. The hepatocytes have glycogenated nuclei, inflammation and variable hepatocellular anisonucleosis. Mallory-Denk bodies (MDB) are present occasionally. Clusters of hepatocytes with granular eosinophilic cytoplasm resulting from increased number of mitochondria are also seen. Copper and hemosiderin pigment deposition within periportal hepatocytes and Kupffer cells is seen.

8. Review of Literature from Bangladesh

A large study from Bangladesh evaluated 941 cryptogenic choric liver disease (CLD) patients who were negative for hepatitis B (HBV) and C viruses HCV), MASLD, alcohol and autoimmune hepatitis to assess if they had been suffering from WD. 24-hour urinary copper was estimated. Of the patients, 239 had WD (24-hour urinary copper >100 µgm/L) and another 239 patients were strongly indicative of having WD (24-hour urinary copper >40 µgm/L). Interestingly KF ring was detected in only 5 patients. The study revealed that WD is no rare condition in Bangladesh, however both patients and general physicians alike were unaware of their underlying liver pathology, pointing to the need for strong clinical suspicion and thorough investigation to establish the cause of cryptogenic CLD [20].

Several interesting cases of WD have also been reported form Bangladesh over the years. The first reported case of adult Wilson' liver disease was that of a 55-years old middle-aged lady who presented with repeated episodes of altered consciousness for 2 years. She also had altered consciousness on admission along with anemia, jaundice, dependent oedema, moderate ascites and palpable spleen. Her plantar response was bilaterally extensor. She had KF ring on Ophthalmic examination. Her serum bilirubin was 5 mg/dL, INR 2.55 and serum albumin 1.8 gm/dL. On abdominal ultrasonography, there was coarse liver, splenomegaly and moderate ascites. Endoscopy of UGIT revealed multiple columns of varices (form 2) and gastric fundal varices. There were δ waves in her electro-encephalogram (EEG). Her serum ceruloplasmin was 156 µ and 24-hour urinary copper was 30 µgm/L. She was treated with D-penicillamine and zinc acetate in addition to supportive measures [21].

The next case was reported by our group. Our patient was an 18-years old, male college student who presented with prodromal features e.g., jaundice, nausea, vomiting and appetite loss. He had tender hepatomegaly. His serum bilirubin was 14.4 mg/dL, ALT 1270 U/L and INR 2.1. His serum ceruloplasmin was 440 mg/ml and 24-hour urinary copper was 1140 µgm/L. He tested negative for HBV, HCV, hepatitis A and E viruses as well as for ANA, ASMA, anti-LKM1 and AMA (M2). He had no KF ring. On whole abdominal ultrasonography, he had hepatomegaly with contracted gall bladder. We diagnosed him as a case of acute WD and treated him with D-penicillamine and zinc along with supportive treatment [2].

There is report of a 24-years old, female college student who had swelling of legs for 1-year, severe generalized weakness for 4 months, high grade fever, vomiting, oliguria, subconjunctival hemorrhage and excessive per-vaginal bleeding. She had severe anemia, mild oedema, hepatosplenomegaly and mild ascites. There was no KF ring. Her serum bilirubin was 1.7 mg/dL, serum ceruloplasmin 23 mg/dL and 24-hour urinary copper was 1416 gm/dL. Abdominal ultrasonography revealed coarse hepatic parenchyma, ascites and dilated portal and splenic veins. Her ascitic fluid was transudative (SAAG 12 gm/L). She tested negative for ANA, HBV and HCV. Her diagnosis was WD related decompensated LC and she received D-penicillamine along with supportive treatment [22].

Recently the case of a 19-years old young female has been reported. The patient had jaundice, fever, neuropsychiatric symptoms and personality changes, including bizarre behavior, initial mutism followed by irrelevant speech as well as oral ulcers, alopecia, arthralgia involving small joints of hands and feet, palpitation and shortness of breath. Her serum bilirubin was 9.4 mg/dL, serum IgG 24.1 gm/L, serum ceruloplasmin 0.2 gm/L and 24-hour urinary copper 204 µgm/L. She tested positive for ANA, anti-dsDNA, anti-smooth muscle, anti-Ro, anti-La, U1-RNP, rheumatoid factor, anticardioloipin, lupus anticoagulant antibodies and direct Coombs test and anti-HEV IgM and her urinalysis revealed red blood cells 8-10/hpf. She tested negative for HBV and HCV and her Complement levels were low. Abdominal ultrasonography revealed mild hepatomegaly and free fluid in sub-hepatic space. Based on Leipzig score (>4) and European Alliance of Associations for Rheumatology (EULAR) criteria, our final diagnosis was WD with Systemic Lupus Erythematosus (SLE) with acute hepatitis E. She was managed with injection methylprednisolone followed by oral prednisolone, oral D-penicillamine and pyridoxine. The patient eventually recovered and was discharged [23].

Another case was also reported recently, who was a 20-years old male from a tea garden in Sylhet. He presented with anemia, jaundice, ascites and splenomegaly for 9 months. Although he was born to non-consanguineous parents, 2 of his elder siblings had been diagnosed with WD. His serum bilirubin was 3.8 mg/dL, INR 50, serum ceruloplasmin 0.010 gm/L and 24-hours urine copper 12.62 µgm/24 hours. He tested negative for HBV and HCV and had KF ring. Ultrasonography revealed coarse hepatic parenchyma, splenomegaly and huge ascites and there were esophageal varices (form 2-3) on endoscopy of UGIT. As the diagnosis of WD was confirmed, he was treated with D-penicillamine and supportive measures [24].

All the cases of WD reported from Bangladesh as well as the original article, which we have reviewed here have important learning points for Hepatologists, Gastroenterologist and Internists alike. They represent the wide spectrum of presentations of WD and thus the challenges in diagnosis. The 3 cases that we have presented here also have a critical angle. In all these 3 cases, WD was co-existent with a much commoner hepatic pathology. Our conventional teaching usually blocks our minds from digging deeper once we are able to attain a diagnosis. Bangladesh is in the intermediate prevalence zone of HBV and we have a high and progressively increasing burden of MASLD in the country. Therefore, in each of these 3 cases, once we had identified the underlying etiology, the diagnosis of WD was much more unlikely than likely. Strong clinical suspicion and evaluation further remain critical for picking up infrequent diseases like WD in regions endemic for HBV and MASLD like ours, as otherwise we will lose some of our patients unknowingly assuming that we were on the right path in their management.

9. Conclusion

WD is a genetic disease resulting from accumulation of excess copper in liver and/or brain. Diagnosis is often challenging due to complex and wide-ranging clinical manifestations. 24-hour urinary copper estimation remains the mainstay of diagnosing WD. If untreated WD is fatal, but the prognosis is excellent with drugs like D-penicillamine, zinc and trientine. Strong clinical suspicion and extensive investigation are vital to attain the diagnosis on many occasions. Besides, patients undergoing treatment should be monitored carefully with Biochemistry, Hematology and urine analysis. Liver transplantation is indicated in end stage Wilson's hepatic disease.

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