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Case Study on Decompensated Liver Disease with Secondary Wilson's Disease & Grade 1 Hepatic Encephalopathy

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KEYWORDS

Wilson disease, Hepatolenticular degeneration, Kayser- Fleischer ring, Hepatoprotectants, copper chelators.

ABSTRACT:

Introduction: Wilson's disease is a rare autosomal recessive disorder of impaired copper excretion that is characterized by the accumulation of copper in many tissues and organs principally in the liver, brain, cornea, and kidneys. It may be present at any age, but majorly between 5 and 35 years.

Case report: A 14 years old male patient was admitted to the hospital with complaints of abdominal distension for the last 6 months. The distension was gradual in onset and progressive in nature. The patient also had complaints of loss of appetite and excessive sleepiness in the morning for 2 weeks. Before admission, the patient was diagnosed with Liver cirrhosis (decompensated), thrombocytopenia and had a history of jaundice 6 months back. Therapeutic and diagnostic tapping, serum ceruloplasmin, serum copper, Anti-LKM, Anti-SMA tests were done.

Conclusion: A practical message is that it should be suspected in young patients presented with unexplained hepatic complications. Early diagnosis and appropriate treatment may help to prevent systemic complications. Adherence to treatment, a low copper diet, and proper follow-up can be helpful in reducing morbidity and mortality.

INTRODUCTION:

Wilson's disease, also called hepatolenticular degeneration, was first described in 1912 by neurologist Kinnear Wilson, who recognized the association of a familial neurological disease with liver disease. It is caused by a disorder of copper metabolism in the liver and is identified as an important cause of acute and chronic liver disease in children. Mutations in the transmembrane protein ATP7B cause impaired hepatic copper excretion, resulting in systemic copper accumulation and toxicity (1). Over the last decade, India has been one of the geographical hot spots for Wilson Disease. The World Health Organization (WHO) has estimated the worldwide prevalence of WD to be 30–100/million. In South India 15–20 new cases of WD were reported every year (2). Wilson's disease, usually presenting with neurological, psychiatric or hepatic manifestations (3). The most important step in the diagnosis of Wilson's disease is consideration in the differential diagnosis. Any unexplained liver or neuropsychiatric disease should be considered in the differential diagnosis (4). Liver Wilson's disease can have similar symptoms as autoimmune hepatitis and non-alcoholic steatohepatitis. Coombs-negative hemolytic anemia, renal tubular dysfunction, pancreatitis, dysrhythmias, rickets, fractures, and amenorrhea are other presenting symptoms. Kayser-Fleischer rings also support Wilson's disease (5).

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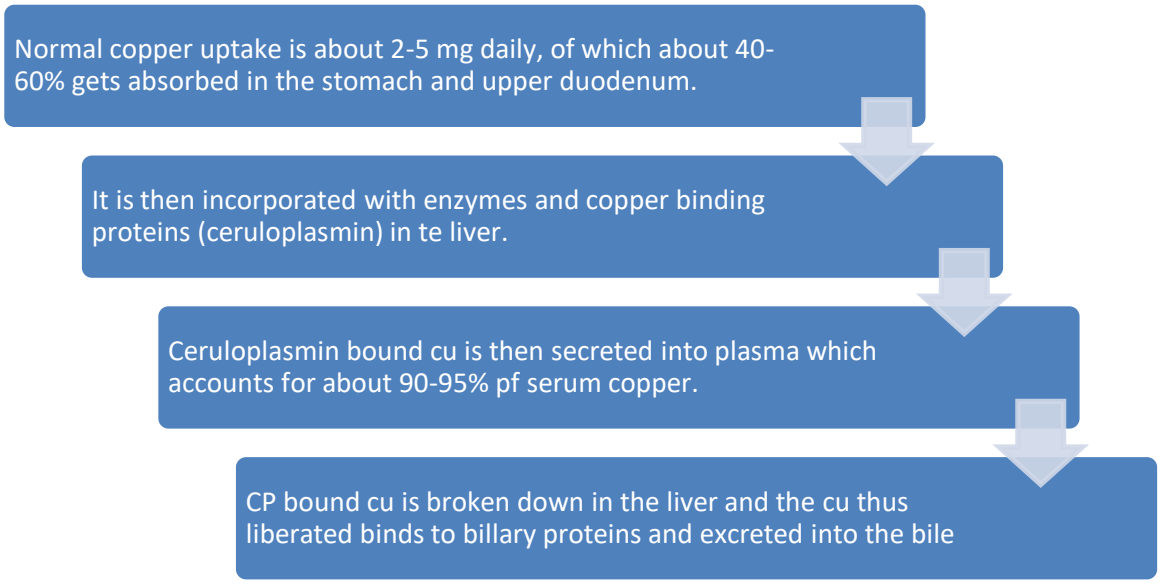
Laboratory findings such as low alkaline phosphatase, aspartate transaminase (AST) higher than alanine transaminase (ALT), and low serum ceruloplasmin are characteristic of Wilson's disease. A 24-hour urine copper collection and liver biopsy with copper quantification are recommended in suspected patients. So far, no single diagnostic test is considered a perfect gold standard.

COPPER METABOLISM AND DEPOSITION:



CASE REPORT

A 14 yrs. an old male patient was admitted to the hospital with complaints of abdominal distension for the last 6 months. The distension was gradual in onset and progressive in nature. The patient also had complaints of loss of appetite and excessive sleepiness in the morning for 2 weeks. Before admission, the patient was diagnosed with Liver cirrhosis (decompensated), thrombocytopenia, and ascites and was on natural medication (Siddha). The patient had a history of jaundice 6 months back. No relevant family history was found. On general examination, the patient was conscious, oriented, afebrile, Icterus, and pale. B/L Pitting edema was seen and was extending up to the thigh. On abdominal examination, diffuse distension, warmth, shifting dullness, and fluid thrill were seen. The portal veins were dilated and the umbilicus was everted. On CNS examination, the B/L pupil was reactive, obeyed commands, and moved all 4 limbs. The patient was provisionally assigned a diagnosis of Decompensated liver disease with portal hypertension and esophageal varices, grade 1 hepatic encephalopathy.

The patient was put on a salt-restricted diet < 2g/day and fluid restriction <1L/day. Diuretics, carvedilol (3.125mg), ursodeoxycholic acid (300mg), and Rifaximin (550mg), Inj.Vitamin K one ampule, Syrup. Lactulose 15ml BD was prescribed and further investigations were carried out. Vitals, weight, abdominal pain and intake/output were monitored and charted every day. CBC, LFT, RFT, Sr. Electrolytes, peripheral smear, Blood grouping and typing, Urine c/e, ECG, 2D ECHO, Therapeutic and diagnostic tapping, serum ceruloplasmin, serum copper, Anti-LKM, Anti-SMA tests were done and the values are given in table.1. Since the patient is young and presented with unexplained liver disease, on suspicion of Wilson's disease a slit lamp examination of the eyes was performed on day 2 of admission and revealed the presence of Kayser Fleischer rings on both eyes. His Ascetic fluid was examined and the following values were noted Albumin-0.3 g/dl, Cl-109 mmol/L, glucose-108 g/dl, LDH- 41U/L, protein-1.0 g/dl. His MELD score was calculated and found to be 31 and his SAAG score was found to be 1.4. His LFT, RFT, and Sr. Electrolytes were abnormal.

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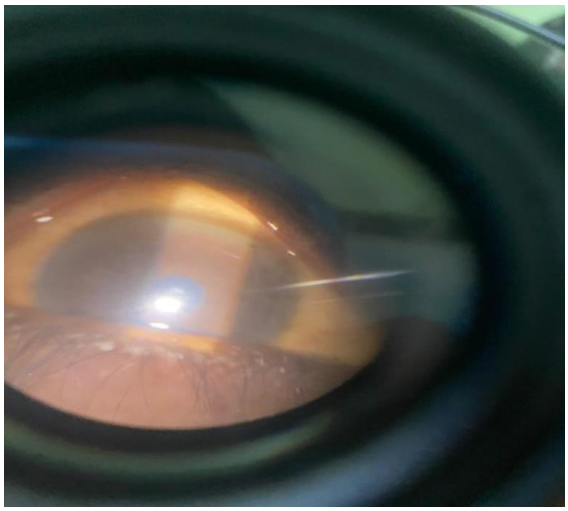

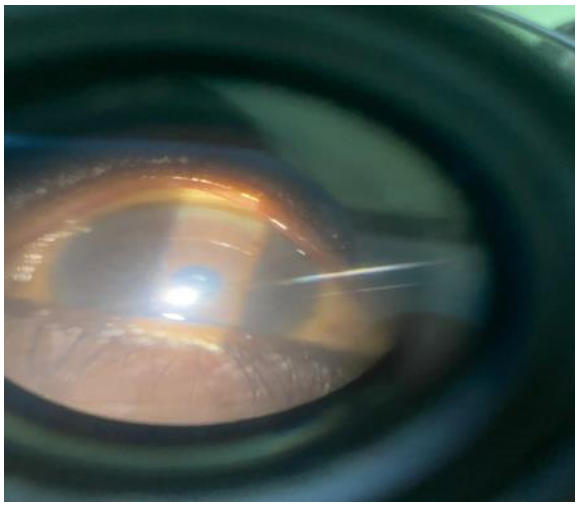

Fig.1. Kayser- Fleischer ring seen with slit lamp

Table.1. Laboratory Investigations

Parameters	**Observed Value**
Hb	10 g/dl
PCV	32%
MCH	34 pg
MCHC	31 g/dl
MCV	108fL
Platelets	63000 cells/cu.mm
PT with INR	42.1 secs/ 3.1
Liver Function Test:	
Total Bilirubin	5.34 mg/dl
Direct bilirubin	3.16 mg/dl
AST	74 U/L
ALT	29 U/L
Total protein	6.0 g/dl
Albumin	1.7 g/dl
Globulin	4.3g/dl
ALP	190U/L
Renal Function Test:	
Serum. Creatinine	0.6 mg/dl
Serum. Urea	6mg/dl
BUN	3mg/dl
Electrolytes:	
Sodium	126 mmol/L
Potassium	3.7 mmol/L
Chloride	105 mmol/L
Bicarbonate	19 mmol/L
Calcium	7.2 mg/dl
Phosphorous	1.3 mg/dl
Magnesium	2.1 mg/dl

Hb- Hemoglobin, PCV-

Serology: HBSAG, HAV, HCV- NON-REACTIVE

Serum Free Copper: Sfr = Sr copper in mg/dl – ceruloplasmin in mg/dl x 3.15 **SFR** = 25.6

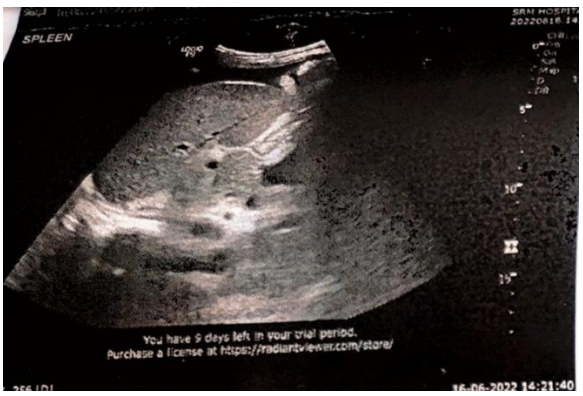

Fig.2. Transverse image of the right lobe of the liver in patient with Wilson disease

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DISCUSSION

Wilson's disease is a rare autosomal recessive disorder of copper excretion, which is characterized by the accumulation of copper in many organs and tissues, mainly in the liver, brain, cornea and kidneys1). It can appear at any age, but most often between the ages of 5 and 35. Diagnosis is very difficult and varies depending on age. Therefore, it is important to suspect Wilson's disease in any child with unexplained liver disease3). It is a progressive disease that could be fatal if left untreated, and early diagnosis is a major challenge. Our patient had yellowish eye color, leg swelling, abdominal distension, and elevated LFT values, indicating liver disease, an early feature of Wilson's disease5). Sonographic examination of the abdomen revealed the presence of hepatomegaly, signs of hepatitis and fibrosis. Neurological features of the disease were also observed in our patient in terms of symptoms such as tremors, incoordination, dystonia, rigidity, difficulties with motor movements and dysarthria. The basal ganglia, cerebellum, and brainstem were common areas affected by copper accumulation in the brain7). It occurs when excess copper builds up in the lenticular area of the brain and usually manifests as difficulty walking, anxiety, mood swings and depression. Another clinical sign is Kayser Fleisher rings, which indicate copper deposits around the cornea in both eyes of the patient. These rings appear red, bright green, or brown, and there are no significant clinical problems associated with the development of these rings and they disappear after treatment8). Treatment for Wilson's disease includes copper chelators such as Penicillamine and zinc. Hepatoprotectors and diuretics were used as supportive measures, where liver transplantation is a life-saving measure for advanced Wilson's disease and was recommended to the patient, but the significance of liver transplantation in severe neurological Wilson's disease is unclear. Thus, Wilson's disease was suspected only after the gradual exclusion of all the above conditions and confirmed by a decrease in ceruloplasmin, the demonstration of Kayser-Fleischer rings and an increase in urinary copper excretion9). The patient was prescribed T.D Penicillamine 200 mg once daily and Zinc 50 mg three times daily along with the previously prescribed medications. The patient was restricted to a low copper diet and strictly advised to avoid chocolate, liver, mushrooms and nuts and his therapy was narrowed down to hepatoprotectors, T. furosemide 20 mg once daily, carvedilol 3.125 mg once daily, D Penicillamine zinc and vitamin K10). The patient was reviewed by a gastroenterologist, who pointed out the need for a liver transplant.

CONCLUSION

Wilson's disease is a rare inherited metabolic disorder. The practical message is that it should be suspected in young patients with unexplained liver complications. Early diagnosis and appropriate treatment can help to prevent systemic complications. Adherence to treatment, a low-copper diet, and proper monitoring can help in reducing morbidity and mortality rates. Liver transplantation may be beneficial in acute liver failure. Siblings of those affected needed screening to prevent manifestations.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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