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Short Communication

Acute Liver Failure Due to Liver Lenticular Degeneration

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DESCRIPTION

Liver lenticular degeneration, also known as Wilson's Disease (WD), is a hereditary disease characterized by abnormal copper metabolism with an incidence of approximately 1/30,000 and 1/100,000. The symptoms of WD are diverse. Wilson's disease with acute liver failure has rapid progress and high mortality [1]. Liver Transplantation (LT) is the ultimate life-saving therapy for patients with irreversible ALF. However, it is difficult to determine the optimal time for a transplant. A patient with acute liver failure who was admitted in a hospital was diagnosed with hepatic lenticular degeneration and undergoing liver transplantation. Cases were reported to deepen the doctor's understanding of the disease diagnosis.

Acute liver failure caused by hepatic lenticular degeneration is a rapidly progressing and seriously life-threatening disease. There is no special treatment, it has been reported in the literature can be achieved by plasma exchange, double plasma adsorption, bloo d purification [2]. Plasmapheresis a commonly used method to support the liver and was recognized by WD in 2013 as a Class I indication for the WD American Association of Specialist Physicians (ASFA). If the above treatments still do not reverse the progression of the disease, liver needs a transplant [3]. Acute liver failure is a rare, inherited autosomal recessive disease of copper metabolism and may be more common where affinity is prevalent. More has been known about the disease after it was first described by Kinnier Wilson as `progressive lenticular degeneration in 1912. Over 500 mutations of the ATP7B gene has been identified with no clear genotype to phenotype correlation. Loss of ATP7B function leads various grades of reduced biliary excretion of copper and reduced incorporation of copper into ceruloplasmin; accumulation and toxicity of copper in the liver, brain and other tissues results in liver toxicity and other myriad manifestations of the disease. The clinical features may vary from asymptomatic state to chronic liver disease, acute liver failure, neuropsychiatric manifestations and hemolytic anemia. Diagnosis is based on a combination of clinical signs, biochemical features, histological findings, and mutation analysis of the ATP7B gene [4]. Geographical differences are precise, and the proportion of children with acute liver failure is imbalanced. Early diagnosis requires high suspicion indicators. Ratios of biochemical indicators for early detection need to be validated in all geographic regions and may not be particularly applicable to children. The need for testing for better biomarkers or early detection of Acute Liver Failure continues. Drugs used in the treatment of Wilson disease include copper chelating agents such as d-Penicillamine, trientine and zinc salt. Untreated Wilson disease uniformly leads to death from liver disease or severe neurological disability [5]. Early recognition and treatment has excellent prognosis. Liver transplantation is indicated in acute liver failure and end stage of liver disease.

Taking over doses of acetaminophen is the most common cause for acute liver failure. By addicting to over doses of prescription medications like antibiotics and anticonvulsants are the major causes for the liver failure. Some viruses like Hepatitis A and Hepatitis B can also cause acute liver disease, but the majority of liver failure cases are due to lenticular degeneration.

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