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Short Notes on Wilson's Disease

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Introduction

Wilson's disease is a rare inherited disorder in which copper accumulates in the liver, brain, and other vital organs. Wilson's disease is most commonly diagnosed between the ages of 5 and 35, but it can affect both children and adults. Copper is essential for the growth of healthy nerves, bones, collagen, and the skin pigment melanin. Copper is normally absorbed from food, and excess is excreted via a substance produced in your liver (bile).However, in people with Wilson's disease, copper is not properly eliminated and instead accumulates, potentially to a life-threatening level. Wilson's disease [1-3] is treatable if detected early, and many people with the disorder live normal lives.

Wilson's disease is present at birth, but symptoms do not appear until copper accumulates in the brain, liver, or another organ. The signs and symptoms of the disease differ depending on which parts of your body are affected. They may consist of Tiredness, a loss of appetite, or abdominal pain. A yellowing of the skin and eye whites (jaundice), Discoloration of the eyes in a golden-brown hue (Kayser-Fleischer rings). Wilson's disease is inherited as an autosomal recessive trait, which means that you must inherit one copy of the defective gene from each parent in order to develop the disease. If you only have one abnormal gene, you will not become ill, but you will be a carrier and can pass the gene on to your children.

About the Study

In children with Hunter syndrome, a variety of neurological complications may exist and continue to develop. Excess fluids in your child's brain can cause a variety of neurological problems. The pressure from these fluids can cause severe headaches, interfere with vision, and alter your child's mental state. In addition, your child may develop a condition in which the membranes that surround the spinal cord thicken and scar [4,5]. This puts pressure on and compresses the upper spinal cord. As a result, your child may experience leg fatigue and gradually weaken and become less physically active.

Wilson's disease is more likely to affect you if your parents or siblings have the condition. Ask your doctor if you should get genetic testing to see if you have Wilson's disease. Diagnosis of the condition as soon as possible improves the chances of successful treatment. Wilson's disease can be fatal if left untreated. Scarring of the liver is one of the most serious complications (cirrhosis). Scar tissue forms in the liver as liver cells attempt to repair the damage caused by excess copper, making it more difficult for the liver to function.

Conclusion

Failure of the liver this can happen suddenly (acute liver failure) or gradually over time. A liver transplant could be an option for treatment. Neurological issues that persist tremors, involuntary muscle movements, clumsy gait, and speech difficulties typically improve with Wilson's disease treatment. However, some people continue to have neurological problems despite treatment. Kidney issues Wilson's disease can cause kidney damage, resulting in problems such as kidney stones and an abnormal number of amino acids excreted in the urine. Psychological issues Personality changes, depression, irritability, bipolar disorder, or psychosis are examples of such symptoms. Problems with blood, these may include the destruction of red blood cells (hemolysis), which results in anemia and jaundice.

References

1. Kreymann, Bernhard, Markus Seige and Ursula Schweigart, et al. "Albumin dialysis: Effective removal of copper in a patient with fulminant Wilson disease and successful bridging to liver transplantation: A new possibility for the elimination of protein-bound toxins." *J Hepatol* 31 (1999): 1080-1085.
2. Jhang, Jeffrey S., Michael L. Schilsky and Jay H. Lefkowitch, et al. "Therapeutic plasmapheresis as a bridge to liver transplantation in fulminant Wilson disease." *J Clin Apher* 22 (2007): 10-14.
3. Kodama, Hiroko, Yoshiko Murata and Taketoshi Iitsuka, et al. "Metabolism of administered triethylene tetramine dihydrochloride in humans." *Life Sci* 61 (1997): 899-907.
4. Silva, Ermelinda E. Santos, Jacques Sarles and Jean Paul Buts, et al. "Successful medical treatment of severely decompensated Wilson disease." *J Pediatr* 128 (1996): 285-287.
5. Schilsky M.L., R.R. Blank, M.J. Czaja and Mark A. Zern, et al. "Hepatocellular copper toxicity and its attenuation by zinc." *J Clin Investig* 84 (1989): 1562-1568.

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