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Wilson's Disease: Unraveling the Complexities of Copper Metabolism

Ferenci Warbi*

Department of Clinical Physiology, University of New Caledonia, France

DESCRIPTION

Wilson's Disease, a rare and hereditary disorder, offers a fascinating glimpse into the intricate workings of the human body's metabolism. Named after Dr. Samuel Alexander Kinnier Wilson, who first described the condition in 1912, this ailment stems from an abnormality in copper metabolism. As copper, a trace element, plays a pivotal role in various bodily functions, Wilson's Disease presents a unique challenge that impacts multiple systems within the body. Copper, an essential mineral, is crucial for the proper functioning of enzymes involved in diverse biochemical processes. These enzymes contribute to the formation of connective tissues, the pigmentation of skin and hair, and the generation of energy in cells. However, maintaining a delicate balance of copper levels is paramount. In Wilson's Disease, a genetic mutation affects the ATP7B gene, which codes for a protein responsible for transporting excess copper out of the liver and into the bile for excretion. Due to this malfunction, copper accumulates in the liver and subsequently overflows into the bloodstream, leading to a cascade of complications. The repercussions of Wilson's Disease extend beyond the liver, encompassing various organs and systems. Hepatic symptoms often emerge, including hepatomegaly (enlarged liver), jaundice (yellowing of the skin and eyes), and abdominal pain. If left untreated, this condition can lead to chronic liver inflammation, cirrhosis, and even liver failure. Furthermore, Wilson's Disease can affect other systems including the eyes, kidneys, and blood. The characteristic Kayser-Fleischer rings, a reddish-brown ring that forms around the iris due to copper deposits, can be a telltale sign. Renal involvement might lead to renal tubular acidosis, which affects the kidney's ability to maintain proper acid-base balance. Haemolytic anaemia, a condition where red blood cells are destroyed prematurely, can also arise. A combination of clinical assessment, blood tests measuring ceruloplasmin (a copper-binding protein), urinary copper excretion, and genetic testing helps establish a definitive diagnosis. Imaging techniques like ultrasound and MRI can provide insights into liver and brain involvement. Once diagnosed, treatment aims to mitigate copper accumulation and manage symptoms. Chelation therapy involves administering medications that bind to excess copper, facilitating its excretion through urine. Penicillamine and trientine are commonly used chelating agents. Zinc therapy is another approach, as high levels of dietary zinc can inhibit copper absorption in the gut. Liver transplantation remains a last resort for individuals with advanced liver damage. Wilson's Disease is inherited in an autosomal recessive manner, meaning that an individual must inherit a mutated copy of the ATP7B gene from both parents to develop the disorder. Carrier parents, who possess one normal and one mutated copy of the gene, often display no symptoms but can pass the mutation to their offspring. Genetic counselling becomes imperative for carriers or families with a history of Wilson's Disease, aiding in informed family planning decisions. Treatment for Wilson's disease involves lifelong management. Doctors typically prescribe medications that help the body excrete excess copper and prevent its absorption from the diet. Penicillamine and trientine are commonly used chelating agents that bind to copper, facilitating its elimination through urine. Zinc supplements are also used to block copper absorption in the intestines. In severe cases where there's irreversible liver damage, a liver transplant might be necessary.

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CONFLICT OF INTEREST

The author states there is no conflict of interest.

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Corresponding author Ferenci Warbi, Department of Clinical Physiology, University of New Caledonia, France, E-mail: w_fa@gmail.com

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