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Insights on Hunter's Syndrome

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Introduction

Hunter's syndrome, also called Mucopolysaccharidosis, rare sex-linked hereditary disorder that varies widely in its severity but is generally characterized by some degree of dwarfism, mental retardation, and deafness. The disease affects only males and makes its first appearance during the first three years of life. Speech and mental development are delayed, the child has frequent respiratory infections, and as the disease progresses a typical constellation of physical signs becomes evident: protuberant abdomen, claw hands, excessive hair growth, coarsening of the face, and retarded growth. The disorder is caused by a deficiency in the enzyme iduronate sulfatase.

About the Study

This deficiency results in a defective chemical breakdown of the mucopolysaccharides, carbohydrates essential in the development of the connective tissues, and a consequent accumulation of mucopolysaccharides in the body, which in turn causes the disease's characteristic mental and physical defects [1-3]. Hunter syndrome occurs when a child inherits a faulty chromosome from his or her mother.

Because of that faulty chromosome, an enzyme required to break down complex sugar molecules is either missing or malfunctioning. Massive amounts of these complex sugar molecules accumulate in the cells, blood, and connective tissues in the absence of this enzyme, causing permanent and progressive damage. history of the family. Hunter syndrome is caused by a faulty chromosome, which a child must inherit in order to develop the disease. Hunter syndrome is an X-linked recessive disease, which means that women carry the defective disease-causing X chromosome and can pass it on to their children. However, mothers are not affected by the disease and are unlikely to be aware that they have this faulty chromosome.

Future Prospective

Hunter syndrome almost always affects men. Because they inherit two X chromosomes, girls are far less likely to develop this disease. If one of the X chromosomes is faulty, the normal X chromosome can provide a working gene. However, if a male's X chromosome is defective, there is no other normal X chromosome to compensate for the problem. Hunter syndrome can cause a variety of complications, depending on the type and severity of the disease. Complications can impact the lungs, heart, joints, connective tissue, as well as the brain and nervous system [4,5].

Breathing becomes difficult due to an enlarged tongue, thickened gums, and thickening of the nasal passages and windpipe (trachea). Chronic ear and sinus infections, respiratory infections, and pneumonia are common in children. Sleep apnea, a condition in which breathing is interrupted intermittently during sleep, is frequently present as a result of airway constriction. Heart tissue thickening can result in improper closing of heart valves. As a result, the heart and other organs do not receive blood as efficiently. These conditions frequently worsen as the disease progresses, eventually leading to heart failure. Tissue thickening can also cause aortic and other blood vessel narrowing. This, in turn, can lead to high blood pressure and pulmonary artery narrowing.

The accumulation of undigested complex sugar molecules in connective tissues causes bone, joint, and ligament abnormalities. This stunts your child's growth, causing pain and physical malformations and making movement difficult. Swelling of joint connective tissues, as well as cartilage and bone abnormalities, cause joint stiffness. When your child is in pain, he or she will most likely move less, which can lead to increased stiffness and pain. Dyostosis multiplex refers to a group of abnormalities that are commonly seen in the bones of people with Hunter syndrome. These abnormalities can cause children to have irregularly shaped vertebrae and spines, ribs, arms, fingers, legs, and pelvises.

Acknowledgement

None.

Conflict of Interest

None.

References

1. Morehead, John M. and David S. Parsons. "Tracheobronchomalacia in Hunter's syndrome." *Int J Pediatr Otorhinolaryngol* 26 (1993): 255-261.
2. Sériès F., Y. Cormier, N. Lampron and J. La Forge. "Influence of lung volume in sleep apnoea." *Thorax* 44 (1989): 52-57.
3. Ginzburg, Alan S., Ergün Önal and Robert M. Aronson, et al. "Successful use of nasal-CPAP for obstructive sleep apnea in Hunter syndrome with diffuse airway involvement." *Chest* 97 (1990): 1496-1498.
4. Mayer P., J.L. Pépin, G. Bettega and D. Veale, et al. "Relationship between body mass index, age and upper airway measurements in snorers and sleep apnoea patients." *Eur Respir J* 9 (1996): 1801-1809.
5. Semenza, Gregg L., and Reed E. Pyeritz. "Respiratory complications of mucopolysaccharide storage disorders." *Med* 67 (1988): 209-219.

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