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Short Notes on Familial Hypercholesterolemia

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Introduction

The way the body processes cholesterol is affected by familial hypercholesterolemia. As a result, people with familial hypercholesterolemia are more likely to develop heart disease and have an early heart attack. The genetic changes that cause familial hypercholesterolemia are passed down through families. Although the condition exists from birth, symptoms may not appear until adulthood. Symptoms usually appear in children who inherit the condition from both parents. If this rare and more severe form is not treated, death usually occurs before the age of 20. A variety of medications and healthy-lifestyle behaviours are used to treat both types of familial hypercholesterolemia. Adults and children with familial hypercholesterolemia have extremely high levels of LDL cholesterol in their blood. LDL cholesterol [1-3] is referred to as "bad" cholesterol because it can accumulate in the artery walls, hardening and narrowing them.

This excess cholesterol can be found in certain areas of the skin, tendons, and around the iris of the eyes: The hands, elbows, and knees are the most common places for cholesterol deposits to form on the skin. They can also appear on the skin around the eyes. Tendons Cholesterol deposits may cause the Achilles tendon, as well as some tendons in the hands, to thicken. Eyes a white or grey ring around the iris of the eye caused by high cholesterol levels is known as corneal arcus. This is more common in older people, but it can also happen in younger people with familial hypercholesterolemia. A gene mutation passed down from one or both parent's causes familial hypercholesterolemia. This condition is inherited by those who have it. This modification prevents the body from eliminating the type of cholesterol that can accumulate in the arteries and cause heart disease. If one or both of your parents have the gene mutation that causes familial hypercholesterolemia, you are more likely to develop it. The majority of people with the condition have only one affected gene.

About the Study

In rare cases, a child may inherit the faulty gene from both parents. This can result in a more severe case of the condition. If one or both of your parents have the gene mutation that causes familial hypercholesterolemia [4,5], you are more likely to develop it. The majority of people with the condition have only one affected gene. In rare cases, a child may inherit the faulty gene from both parents. This can result in a more severe case of the condition. You are more likely to develop familial hypercholesterolemia if one or both of your parents

have the gene mutation that causes it. The majority of those affected have only one affected gene. A child may inherit the faulty gene from both parents in rare cases. As a result, the condition may become more severe. A thorough family history is essential in diagnosing familial hypercholesterolemia. Doctors will want to know if your siblings, parents, aunts, uncles, or grandparents have ever had high cholesterol or heart disease, especially when you were a child. Doctors usually look for cholesterol deposits in the skin around the hands, knees, elbows, and eyes during a physical exam.

Conclusion

Tendons in the heel and hand may thicken, and a grey or white ring around the iris of the eye may form. Although a genetic test can confirm familial hypercholesterolemia, it is not always required. A genetic test, on the other hand, can help determine whether other family members are also at risk. Each child has a 50% chance of inheriting familial hypercholesterolemia if one parent has it. Inheriting the faulty gene from both parents can result in a more rare and severe form of the disease. If you are diagnosed with familial hypercholesterolemia, your first-degree relatives, such as siblings, parents, and children, should be tested for the disorder as well. This will allow treatment to begin as soon as possible, if necessary.

References

- Quintão E.D.E.R, Scott M. Grundy, and Edward H. Ahrens Jr. "Effects of dietary cholesterol on the regulation of total body cholesterol in man." J Lipid Res 12 (1971): 233-247.
- Scott P.J. and Christine C. Winterbourn. "Low-density lipoprotein accumulation in actively growing xanthomas." J Atheroscler Res 7 (1967): 207-223.
- Grundy, Scott M., E.H. Ahrens and Gerald Salen. "Interruption of the enterohepatic circulation of bile acids in man: Comparative effects of cholestyramine and ileal exclusion on cholesterol metabolism." J lab clin med 78 (1971): 94-121.
- Lewis, B., and N.B. Myant. "Studies in the metabolism of cholesterol in subjects with normal plasma cholesterol levels and in patients with essential hypercholesterolaemia." *Clin Sci* 32 (1967): 201-213.
- Langer, Terry, Warren Strober and Robert I. Levy. "The metabolism of low density lipoprotein in familial type II hyperlipoproteinemia." J Clin Investig 51 (1972): 1528-1536.

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