Brief Report
Volume 11:3, 2022

Journal of Metabolic Syndrome

ISSN: 2167-0943 Open Access

Insights on Hereditary Hemochromatosis

Lisa Andrew*

Department of Molecular Biology, University of Helsinki, Finland

Introduction

Hereditary hemochromatosis causes your body to absorb an abnormally large amount of iron from the food you eat. Excess iron is stored in your organs, particularly the liver, heart, and pancreas. Too much iron can cause life-threatening conditions like liver disease, heart disease, and diabetes. The genes that cause hemochromatosis are inherited, but only a small percentage of people who have the genes experience serious complications. Hereditary hemochromatosis [1-3] symptoms usually appear in middle age. Blood is removed from your body on a regular basis as part of treatment. Because red blood cells contain a large portion of the body's iron, this treatment lowers iron levels.

About the Study

Hereditary hemochromatosis manifests itself at birth. However, most people do not notice signs and symptoms until later in life, usually after the age of 40 in men and 60 in women. Women are more likely to develop symptoms after menopause because they no longer lose iron through menstruation or pregnancy. Hereditary hemochromatosis is a difficult condition to diagnose. Early symptoms like stiff joints and fatigue could be caused by something other than hemochromatosis. Many people with the disease have no symptoms other than elevated iron levels in their blood. Hemochromatosis can be detected through abnormal blood tests performed for other reasons or through screening of family members of people who have the disease.

Future Prospective

Saturation of serum transferrin this test determines the amount of iron bound to a protein (transferrin) in your blood that transports iron. Saturation levels of transferrin greater than 45 percent are considered excessive. Ferritin in the blood this test determines how much iron is stored in your liver. If your serum transferrin saturation test results are higher than expected, your doctor will check your serum ferritin. Tests for liver function these tests can aid in the detection of liver damage [4,5].

MRI an MRI is a quick and painless way to determine the extent of iron overload in your liver. Examining for gene mutations if you have high levels of iron in your blood, you should have your DNA tested for mutations in the HFE gene. If you're thinking about getting genetic testing for hemochromatosis, talk to your doctor or a genetic counsellor about the benefits and drawbacks. Taking a liver tissue sample for testing (liver biopsy) if liver damage is suspected, your doctor may extract a sample of liver tissue with a thin needle.

Doctors can safely and effectively treat hereditary hemochromatosis by removing blood from your body on a regular basis (phlebotomy), just as if you were donating blood. The goal of phlebotomy is to normalise your iron levels. The amount of blood removed and the frequency with which it is removed are determined by your age, overall health, and the severity of iron overload. Initial treatment plan initially, you may have a pint (approximately 470 millilitres) of blood drawn once or twice a week, usually in a hospital or at your doctor's office. A needle is inserted into a vein in your arm while you are reclined in a chair. Blood flows from the needle into a tube connected to a blood bag.

Treatment for hereditary hemochromatosis can help relieve symptoms such as fatigue, abdominal pain, and skin darkening. It can aid in the prevention of serious complications like liver disease, heart disease, and diabetes. If you already have one of these conditions, phlebotomy may help to slow or even reverse its progression. Phlebotomy will not cure cirrhosis or joint pain, but it will help to slow its progression. If you have cirrhosis, your doctor may advise you to get screened for liver cancer on a regular basis. This is usually followed by an abdominal ultrasound and a CT scan.

Conflict of Interest

None.

Acknowledgement

None.

References

1. Fasouliotis, Sozos J. and Joseph G. Schenker. "BRCA1 and BRCA2 gene mutations: decision-making dilemmas concerning testing and management." *Obstet Gynecol Surv* 55 (2000): 373-384.
2. Beutler and Ernest. "The significance of the 187G (H63D) mutation in hemochromatosis." *Am J Hum Genet* 61 (1997): 762.
3. Barton, James C. and Luigi F. Bertoli. "Hemochromatosis: The genetic disorder of the twenty–first century." *Nat Med* 2 (1996): 394-395.
4. Yang, Quanhe, Sharon M. McDonnell and Muin J. Khoury, et al. "Hemochromatosis-associated mortality in the United States from 1979 to 1992: An analysis of multiple-cause mortality data." *Ann Intern Med* 129 (1998): 946-953.
5. Lubin, Ira M., N. Alice Yamada and Rachel M. Stansel, et al. "HFE genotyping using multiplex allele-specific polymerase chain reaction and capillary electrophoresis." *Arch Pathol Lab Med* 123 (1999): 1177-1181.

***Address for Correspondence:** Lisa Andrew, Department of Molecular Biology, University of Helsinki, Finland, E-mail: LisaAndrew99@gmail.com*

Received: 02-Mar-2022, Manuscript No. jms-22-62746; **Editor assigned:** 04-Mar-2022, Pre QC No. P-62746; **Reviewed:** 18-Mar-2022, QC No. Q-62746; **Revised:** 23-Mar-2022, Manuscript No. R-62746; **Published:** 30-Mar-2022, DOI:10.37421/jms.2022.11.268.

How to cite this article: Andrew, Lisa. "Insights on Hereditary Hemochromatosis." J Metabolic Synd 11 (2022): 268.