

Comparison of PON1 activity generated by HDL in coronary heart disease (CHD) patients and healthy individuals

Ms Deepa Molluru, Research Scholar, Malwanchal University

Prof. Dr Shreya Nigoskar, Research Supervisor, Malwanchal University

Introduction

Coronary heart disease, also known as CHD, is becoming both more common and more severe in countries all over the world. Coronary heart disease, also known as CHD, is the main cause of mortality in Iran. Hypertension and coronary heart disease are typically associated with elevated levels of low-density lipoprotein (LDL), total cholesterol (TC), and triglycerides (TG) in the blood. On the other hand, death from coronary heart disease (also known as CHD) relates to low levels of high-density lipoprotein (HDL). In coronary heart disease (CHD), alterations in lipoproteins have been shown to contribute to the development of both microvascular and macrovascular issues. This is a well-established fact.

Patients diagnosed with coronary heart disease frequently suffer from dyslipidemia, which is characterised by increased levels of triglycerides and cholesterol and decreasing levels of good cholesterol (HDL). Having a ratio of triglycerides to high-density lipoprotein cholesterol (HDL-C) that is more than 4.5 is another important factor in the development of coronary heart disease. Those who suffer from dyslipidemia have a significantly increased likelihood of developing coronary heart disease (CHD), as well as a higher chance of dying from CHD-related causes. Coronary heart disease (CHD) risks and death rates can be kept at more manageable levels with regular monitoring and management of lipid profiles and use of hypolipidemic medicines.

It is thought that the fundamental factor in the action of HDL-antioxidant C is an enzyme that is attached to HDL-C and is known as human serum paraoxonase 1 (PON1). PON1 is involved in the detoxification process in atherosclerotic circumstances. This process is part of detoxification. PON1 and HDL have been established in multiple studies to provide protection against the oxidative modification of LDL. PON1 has been shown in additional studies to be capable of inhibiting the accumulation of lipid peroxide on LDL both in vitro and in vivo. A low PON1 activity level in the blood has been related not only to familial

hypercholesterolemia but also to diseases that speed up the atherogenesis process. Paraoxonase 1 possesses antiatherogenic qualities that are beneficial to lipoprotein particles. These properties include the ability to neutralise free radicals, hydrolyze oxidised cholesteryl esters and phosphatidylcholine core aldehydes, and degrade hydrogen peroxide. Patients with coronary heart disease (CHD) need to pay close attention to how their heart problems change over time, which can be seen in their PON1 activity.

As a consequence of this, we hypothesised that coronary heart disease would be linked to lower blood levels of PON1 activity and HDL, in addition to higher levels of cholesterol, TGs, and atherogenic indices. The association between PON1 activity and HDL and atherogenic indices in CHD has only been the topic of a limited number of studies. This study's objectives were to investigate the association between PON1 activity and HDL and atherogenic indices in CHD and assess the current state of PON1 activity, lipid profiles, and atherogenic indices.

Methodology

This research was carried out at selected cardiac hospitals in Indore on a total of one hundred CHD patients and one hundred healthy controls. The Institutional Ethics Committee gave their blessing to the research project. In the patients' native language, written informed consent was collected from them before the study began. This is a study that is both observational and cross-sectional. The ages of the participants ranged from 40 to 60 years. Patients with coronary heart disease were diagnosed with the condition using coronary angiography. Following a fast of 12 hours that lasted all night, 10 millilitres of whole blood were obtained through vena puncture in the morning 7 am. The analysis of the data was done with unpaired Student's t tests. With the help of Pearson's correlation analysis, we were able to determine the correlation coefficients. Throughout the statistical analysis process, the software package for statistical analysis version 21 (SPSS) for Windows was utilised. It was determined to be statistically significant if the P value was less than 0.05.

Results

Patients with coronary heart disease had significantly higher concentrations of fasting blood glucose, triglycerides, total cholesterol, and very low density lipoprotein (VLDL), whereas patients with CHD had significantly lower concentrations of HDL and PON1 activity than controls. Positive correlations were found between paraoxonase 1 activity and HDL ($r = 0.224$, $p = 0.039$) and negative correlations were found with the atherogenic coefficient ($r = -0.079$, $p = 0.31$), the cardiac risk ratio 1 ($r = -0.078$, $p = 0.31$, Fig. 3), and the cardiac risk ratio 2 ($r = -0.41$, $p = 0.37$). The findings of this study that were most pertinent were significant increases in serum levels of fasting blood glucose, total cholesterol, very low density lipoprotein, low density lipoprotein, atherogenic index, atherogenic coefficient, and cardiac risk ratios 1 and 2 in DHC. According to a number of studies, people who have coronary heart disease have significantly higher levels of total cholesterol, triglycerides, LDL concentrations, and LDL/HDL ratios than healthy people do. Numerous studies have pointed to total cholesterol and LDL as two of the most important contributors to the development of atherosclerotic vascular diseases. In addition, research conducted in the field of epidemiology has uncovered a connection between LDL and CHD. It has also been demonstrated that statins and other lipid-lowering medications lower the risk of cardiovascular diseases. Additionally, it has been demonstrated that the risk of coronary heart disease is inversely related to HDL-C concentration. The atherogenic indices, the atherogenic coefficient, and the cardiac risk ratio are all powerful indicators of the risk of heart disease. The higher the value, the higher the risk of developing coronary heart disease (CHD), and vice versa. Atherogenic indices, atherogenic coefficients, and cardiac risk ratios have all been shown in a number of studies to be major risk factors for atherosclerotic vascular disease and the complications associated with it. The findings of our research are consistent with those of other researchers, who found that individuals with coronary heart disease have higher levels of total cholesterol, triglycerides, and LDL concentrations, as well as higher atherogenic indices, atherogenic coefficients, and cardiac risk ratios than healthy individuals.

As a result, lowering LDL cholesterol levels and atherogenic indices have been suggested as possible ways to lower the risk of coronary heart disease (CHD).

Conclusion

In conclusion, we discovered significantly lower levels of HDL-C and PON1 activity and significantly higher levels of fasting blood glucose, total cholesterol, triacylglycerol, very low-density lipoprotein, low density lipoprotein, atherogenic index, and cardiac risk ratios in CHD patients. These findings lend credence to the hypothesis that low PON1 activity is linked to low HDL concentration, which is a characteristic of coronary heart disease (CHD). Based on our findings, it appears that an early step in the development of atherosclerotic heart disease in CHD may involve a decrease in PON1 activity and an increase in HDL concentration, as well as an increase in FBG, TG, TC, VLDL, and LDL concentrations, as well as an increase in the atherogenic index, the atherogenic coefficient, and cardiac risk ratios. However, long-term clinical studies are required to shed light on the pathophysiological role that serum PON1 activity and lipid profile play in coronary heart disease (CHD), as well as the correlation between PON1 activity and HDL and the atherogenic index.

Reference

1. Schürks M, Rist PM, Bigal ME, Buring JE, Lipton RB, Kurth T. Migraine and cardiovascular disease: systematic review and meta-analysis. *BMJ* 2009;339:b3914.
2. Silva FA, Rueda-Clausen CF, Silva SY, Zarruk JG, Guzmán JC, Morillo CA, Vesga B, Pradilla G, Flórez M, López-Jaramillo P. Endothelial function in patients with migraine during the interictal period. *Headache* 2007;47:45-51.
3. Hamed SA. The vascular risk associations with migraine: relation to migraine susceptibility and progression. *Atherosclerosis* 2009;205:15-22.

4. Bigal ME, Kurth T, Santanello N, Buse D, Golden W, Robbins M, Lipton RB. Migraine and cardiovascular disease: a population-based study. *Neurology* 2010;74:628-635.
5. Marmura MJ. Systemic abnormalities in migraine: what comes first? *Neurologist* 2009;15:53-54.
6. Kurth T. Migraine and ischaemic vascular events. *Cephalalgia* 2007;27:965-975.
7. Sacco S, Ripa P, Grassi D, Pistoia F, Ornello R, Carolei A, Kurth T. Peripheral vascular dysfunction in migraine: a review. *J Headache Pain* 2013;14:80.
8. Aviram M, Rosenblat M. Paraoxonases and cardiovascular diseases: pharmacological and nutritional influences. *Curr Opin Lipidol* 2005;16:393-399.
9. Gupta N, Gill K, Singh S. Paraoxonases: structure, gene polymorphism & role in coronary artery disease. *Indian J Med Res* 2009;130:361-368.
10. Gan KN, Smolen A, Eckerson HW, La Du BN. Purification of human serum paraoxonase/arylesterase. Evidence for one esterase catalyzing both activities. *Drug Metab Dispos* 1991;19:100-106.
11. Mackness MI, Harty D, Bhatnagar D, Winocour PH, Arrol S, Ishola M, Durrington PN. Serum paraoxonase activity in familial hypercholesterolaemia and insulin-dependent diabetes mellitus. *Atherosclerosis* 1991;86:193-199.
12. Sentí M, Tomás M, Fitó M, Weinbrenner T, Covas MI, Sala J, Masiá R, Marrugat J. Antioxidant paraoxonase 1 activity in the metabolic syndrome. *J Clin Endocrinol Metab* 2003;88:5422-5426.
13. Headache Classification Subcommittee of the International Headache Society (IHS). The International Classification of Headache Disorders, 3rd edition

(betaversion). Cephalalgia 2013;33:629-808.

14. Stewart WF, Lipton RB, Dowson AJ, Sawyer J. Development and testing of the Migraine Disability Assessment (MIDAS) Questionnaire to assess headache-related disability. Neurology 2001;56(Suppl 1):20-28