Available online at www.elixirpublishers.com (Elixir International Journal)

Hormones and Signaling

Elixir Hormo. & Signal. 77 (2014) 29304-29306

ISSN: 2229-712X

Lipid and lipoprotein profile in liver diseases

Bamigbowu E. Olugbenga[1,*], Adegoke O.Adebayo[2], Nwagu Chinyere[3] and George-Opuda M. Ibitoroko[2]

[1]Department of Chemical Pathology, University of Port Harcourt, Nigeria.

[2]Department of Medical Laboratory Science, Rivers State University of Science and Technology, Port Harcourt, Nigeria.

[3]Department of Medical Laboratory Science, Imo State University, Owerri, Nigeria.

ARTICLE INFO

Article history:

Received: 7 September 2014;

Received in revised form:

10 December 2014;

Accepted: 17 December 2014;

Keywords

Lipid,

Lipoprotein,

Liver, Disease.

ABSTRACT

The study was carried out on 55 subjects made up of 27 apparently healthy individuals and 28 subjects who had liver disease. Their lipid and lipoprotein profiles were estimated using standard methods. The result showed that total cholesterol was significantly reduced from 4.71+1.44mmol/l in control to 3.37+1.46 in hepatic disease (P<0.05). Also HDL and LDL cholesterol were significantly reduced (P<0.05) from 0.89+0.37mmol/l and 3.25+1.54mmol/l respectively to 0.31+0.11mmol/l and 2.00+1.30mmol/l while triglycerides significantly increased (P<0.05) from 1.29+0.80mmol/l in control to 2.50+0.89mmol/l in hepatic disease. The result of the study showed that liver diseases can cause lipid dysfunction.

Introduction

The liver is the largest major organ of the human body. It is the site of most metabolic activities that are required for the sustenance of the human life (Gorman, 1992, Rifai *et al*; 2001). The metabolism of lipid and lipoprotein is one of such fundamental functions of the livers. The fate of lipids and lipoprotein in disease conditions of the livers in adults has necessitated this project.

The natural fats or lipids that are weakly polar and as such hydrophobic in nature are normally stored in tissues largely in water free state. Such serve as reservoirs of energy, available in terms of restricted nutrition for the operation of the numerous organs.

Lipid in diet serves the purpose of availing the human body with The lipid soluble vitamins (vitamin A,D,E, and K) and hormones (Murray *et al*, 2000) which are refined for optimal growth and maintenance of tissue (Whyte *et al*, 1978), (Baron, 1973). It is the most concentrated source of energy over twice as many calories of carbohydrate and protein (Whyte *et al*, 1978), (Baron, 1973), (Murray *et al*, 2000). It helps insulate the body against excessive heat loss to the environment and against mechanical trauma as can be seen during the intrauterine life of the mammalian feotus in relation to the function of depot of lipid during adult life As electrical insulators allowing rapid propagation of depolarization wave along myelinated nerves (Murray *et al*, 2000).

The two most commonly known lipoprotein are low – density lipoprotein (LDP) called 'bad cholesterol' and high density lipoprotein (HDL) known as 'good cholesterol' while the former contributes to the formation of plague build up in the arteries, known as atherosclerosis, the later helps to remove cholesterol from the blood, preventing the fatty build up and formation of plague (UVHS 2004).

Significant relationship between high density lipoprotein cholesterol (HDLP) and biopsy- documented liver disease showed that HDL decreased strikingly and significantly in acute alcohol hepatitis and in acute viral hepatitis, while there was moderate decrease in HDL with inactive alcohol liver disease and chronic active hepatitis (UVHS 2004).Low HDL cholesterol along with increased TGs and uric acid had been reported in most biopsy proven nonalcoholic fatty liver disease patients with central fat accumulation (Marceline *et al*, 2001).

Observation and experimental studies have shown that subtle membrane changes are sufficient to allow the passage of intracellular enzymes to the extracellular space. Cell damage increases membrane permeability causing cytisilic enzyme to spill into the sinusoid and from there, into the peripheral blood.

The aim of this study is to determine lipid profile (Cholesterol, Triglyceride, HDL Cholesterol and LDL Cholesterol) in liver dysfunction caused by hepatitis, cirrohosis and cancer of the liver.

Materials and Methods

Subjects

Fifty Five (55) subjects made up of twenty seven (27) apparently healthy individuals, used as control and twenty eight (28) subjects with liver Dysfunction caused by hepatitis, cirrohosis and cancer of the liver. The control were chosen from those patients with primarily low levels of hepatic enzymes activities (Aspartate Transaminase (AST), Alanine Transpminase and Alkaline phosphates (ALP))with bilirubin status considered secondarily to be within the normal range of 5 – 17 µmol/L for the total bilirubin and < 8.5µmol for the conjugate bilirubin.

Commercially prepared AST, ALT, Bilirubin Cholesterol, triglycerides, and HDL precipitant were obtained from Randox Diagnostics, London while QCA produced ALP reagent was obtained from QCA Spain.

Biochemical Studies: The cholesterol is determined after enzymatic hydrolysis and oxidation. The indicator quinoneimine is formed from hydrogen peroxide and 4-aminoantipyrine in the presence of phenol and peroxides (Allain et al 1974).

Ten microlitre ($10^{\mu l}$) of sample, control, standard and distilled water was pipette into respective test tube then $1000^{\mu l}$ of cholesterol working reagent was added. It was mixed and incubated for 5 minutes at 37^0C. The absorbance of the sample was measured against the reagent blank at 520nm. The concentration of sample was calculated using the absorbance of

Tele:

E-mail addresses: bayoadeghq@yahoo.com

sample against absorbance of standard multiplied by concentration of standard.

The triglycerides are determined after enzymatic hydrolysis with lipases. The indicator is a quinoneimine formed from hydrogen peroxide, 4-aminophenazone and 4-chlorophenol under the catalytic influence of peroxidase (Buccolo and David 1973).

Ten microlitre (10) μl of sample, control, standard and distilled water was pipetted into respective test tube then 1000 μl of triglyceride reagent was added. It was mixed and incubated for 5 minutes at 37^0C. The absorbance of the sample was measured against the reagent blank at 520nm. The concentration of sample was calculated using the absorbance of sample against absorbance of standard multiplied by concentration of standard.

Low density lipoproteins (LDL and VLDL) and chylomicron fractions are precipitated quantitatively by the addition of phosphotungstic in the presence of magnesium ions. After centrifugation, the cholesterol concentration in the HDL (high density lipoprotein) fraction, which remains in the supernatant, was determined.

Five hundred (500) μl of sample, control standard and distilled water was added into respective test tubes, 1000 μl of precipitant was added into all the tubes. It was mixed and allowed to stand for 10 minutes at room temperature. It was centrifuged for 2 minutes at 12,000 rpm. Then 10 μl of supernatant from control, standard and distilled water was added into their respective test tubes and cholesterol concentration of supernatant was determined as shown above by method of Allain et al (1974).

LDL-cholesterol was calculated using the formula of Friedwald et al (1972) as shown below

LDL-cholesterol (Mmol/L)=Total cholesterol (Mmol/L)-(HDLC (Mmol/L)+TG/2.22)(Mmol/L).

Statical Analysis: The data generated were subjected to statistical analysis including the mean (x), standard deviation (SD) and student's t – test.

Results

The result of the study showed that total cholesterol concentration increase from 3.37±1.46mmol/l in hepatic disease state to 4.71±1.44mmol/l in control (P<0.05).Also triglyceride decreased from 2.50±0.89mmol/l in hepatic diseased state to 1.29±0.80mmol/l in control (P<0.05). The HDL Cholesterol concentration was 0.31±0.11mmol/l in hepatic disease state while it was 0.89±0.37mmol/l in the control (P<0.05). The LDL concentration was 2.00±1.37mmol/l in hepatic disease state but 3.25±1.54mmol/l in the control (P<0.05) as shown in table 1 below.

Table 1: Lipid profile of subjects with Hepatic disease

Parameter (Mmol/l)	Hepatic Diseased	Control	T	P Value
Total Cholesterol	3.37±1.46	4.71±1.44	12.18	P< 0.05
Triglyceride	2.50±0.89	1.29±0.80	15.00	P< 0.05
HDL Cholesterol	0.31±0.11	0.89±0.37	14.00	P< 0.05
LDL Cholesterol	2.00±1.37	3.25±1.54	6.19	P< 0.05

Mean ±SD

Discussion

It is general reported by several authors that the liver is the centre for metabolic activities that are vital for the sustenance of life of an individual. Being centrally placed place in the body, the liver receives blood supply mainly from the hepatic portal vein and the hepatic artery. The blood contains absorbed metabolites including the vitamins/minerals and toxicants from the gut for processing. This presupposes the fact that a lot of biochemical reactions go on at the same time, in the liver. Some of, which are integrated, others are of independent pathways. All these are pointing and buttressing that fact that the liver is a large, tough and resilient organ of the body with an in numerable talent (Bishop *et al*, 1999).

To have the knowledge of the fate of lipid and lipoproteins in the liver when it is said to be malfunctioning or when hepatocellular damage had occurred, during which the endogenous hepatic enzymes (AST, ALT, ALP as well as γ-glutamate transferase) level become high in the plasma (Tolman, *et al*, 2001, Huruk *et al*, 1999) was the driving desire that prompted this research work.

The project has summarily revealed that in cases of hepatic cell damage, all lipoproteins, total cholesterol (TC), HDL and LDL were significantly decreased while Triglyceride level significantly increased. This is in agreement with the reported findings of Maechesini *et al;* (2001) and Nduka, (1999) and Rifai et al; (2001).

Marchesini *et al*, (2001) and Nduka (1999) had earlier reported that plasma Uric acid level increased along with triglycerides in nonalcoholic fatty liver disease. This is also consistent with the report of this project. The findings of this study are also in agreement with report of UVHS (2004). Baron *et al*., (1973) is among those that pointed out that there exist a decreased level of plasma ester cholesterol when there is parenchyma liver cell damage, which report is also consistent with the finding of this project.

The increase in triglyceride level in the plasma during hepatocellular damage may not be unconnected with excessive mobilization of fatty acids especially from the adipose tissue – (where they are stored) that were subsequently transformed into endogenous triglyceride. The essence of mobilizing the fatty acids from the storage – the adipose tissue was primarily to produce cellular energy in the fasting state (loss of appetite) in the abnormal patients. This is the view of Whyte *et al* 1978 and Gorman *et al.,* 1999, which is in agreement with the reported findings of this project.

Conclusion

The study has shown that hepatocellular damage causes changes in the profile of lipid and lipoproteins such that the Triglycerides (TGs) significantly increased while the total cholesterol (TC), the High- density lipoprotein (HDL) and the low-density lipoproteins (LDL) significantly decreased.

References

Baron D.N (1973) 'Lipids' A short textbook of chemical Pathology 3rd Ed 60-70.

Gorman, L.S; (1992) 'Lipoproteins' Clinical chemistry 2nd Ed, by Bishop ML & Co. 353-371

Huruk, B.L; and Kerfetz; R.G; (1992) 'Enzymes in Hepatic disorder' clinical chemistry 2nd Ed. By Bishop ML & Co. 241

Marchesini G; Brizi, M; Blanchi G Tomassitti S; Bagianesi E; Lenz, M: McCullough, Aj; Natale S; Forlani CS, Melchioda N; (2001) 'Non alcoholic

Murray, RK; Coranner, DK, Mayes, P.A, Rodwell, V.W, (2000) 'Lipid of physiologic significance' Harpers' Biochemistry 25th Ed. 160-177 and 790

Nduka, N (1999) 'Chemical Toxicology' Clinical Biochemistry for Students of Pathology 196-195

Rifai, N; Bachori K, P.S and Albers, J.J; (2001) 'Lipid, Lipoproteins, and Apolipoproteins', in Fundamentals of Clinical Chemistry 5th Ed, by Tietz, W.N. 462-493

Tolman, K G; and Rej R; (2001) 'Liver function', Fundamentals of Clinical chemistry, 5th Ed. By Tietz W.N. 747-770.

University of Virginia health System (UVHS) (2004) 'Cholesterol, LDL, HDL and Triglycerides' UV. Health topics A to Z (4) 1-4

Whyte, A; Handler, P. Smith, E; Hell, R; Lehman, I, R (1978) Lipid metabolism I.II and III).Principle of Biochemistry 6th Ed 38-69 and 568-647.