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Ultrasonographic Findings of Selected Liver Parameters and Their Correlation to Lipidaemia in a Nigerian Population

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Abstract

Introduction: This study was conducted in the University of Port Harcourt Teaching Hospital, with the sample analysis conducted in HMG Hospital private laboratory in Rivers State. **Methodology:** A random sampling technique was employed to select the respondents, while the Taro-Yamene formula was used to calculate the sample size and data analysed with SPSS version 20. **Results:** The respondents were mainly aged 30 - 39 years, 12 (40.00%), mainly females, 20 (66.67%) and obese, 16 (53.33%). They were also mainly Christians, 25 (83.33%), of Ijaw descent 20 (66.67%) and civil/public servants, 13 (43.33%). The total cholesterol was the highest, 18 (60.00%), normal for triglyceride, 24 (80.00%), low for high density lipoprotein cholesterol, 22 (73.33%) and high for low density lipoprotein cholesterol, 14 (46.67%). Maximum liver span was statistically significant to triglyceride concentration; p-value (0.001) but not for total cholesterol; p-value (0.084), high density lipoprotein cholesterol; p-value (0.477) and low density lipoprotein cholesterol; p-value (0.317). **Conclusion:** Liver span is a predictive tool for the probable diagnosis of dyslipidaemia.

Keywords

Liver, Triglycerides, Cholesterol, Lipoprotein, Lipid

1. Introduction

Life is associated with several medical conditions, some of which arise from either the food we consume, genetics, infections, our environment or lifestyle that affects the liver. Hyperlipidaemia, also called lipidaemia (increased presence of lipids in circulation), is one of the cause of liver disease and results from malfunction in the liver. It is occasionally associated with fatty liver, affects between

25% - 37% of healthy adult [1] and can arise from overweight, hyperglycemia, hyperuricemia and chronic alcoholism [2].

The liver consists of two major parts (right and left), divided by the falciform ligament to form four lobes (left, right, caudate, and quadrate lobes) when viewed from the top and below respectively. Other anatomical landmarks include the ligamentum venosum, ligamentum teres (divides the left lobe in two sections) and the portahepatis (divides left lobe into four segments). The organ also has a diaphragmatic surface (resting on/overlaid by the diaphragm and covered by a thin, double-layered membrane, peritoneum) and visceral/inferior surface, and several impressions (colic, renal, suprarenal, duodenal and gastric impressions).

Microscopically, each liver lobe comprises of hepatic lobules (roughly hexagonal plates of hepatocytes radiating from a central vein). A distinctive component of the lobule is the portal triad, consisting of five structures: a branch of the hepatic artery, a branch of the hepatic portal vein, bile duct, lymphatic vessels and a branch of the vagus nerve. Between the hepatocyte plates are liver sinusoids (enlarged portal triad capillaries receive blood from hepatic portal vein and hepatic artery enters and drains into the central vein).

Lipids are biological or synthetic substances that are insoluble in water, but for alcohol, ether or chloroform, and hyperlipidaemia is mainly attributed to metabolic malfunction of the liver, making the liver churn out more lipids into the circulation than required for cellular homeostasis. They are of plant or animal origin [3]. The clinical importance of hyperlipidemias is associated with the pathologic processes emanating to, notably, cardiovascular disease conditions, such as coronary heart disease. Among the functions of the liver is drug, hormone and bilirubin metabolism, as well as, protein catabolism to form urea and other metabolic processes [4]. It is thus, reported that hyperlipidemia could arise from biliary obstruction, diabetes mellitus, hypothyroidism and nephrotic syndrome [5]. It is also reported that hyperlipidaemia can be due to intrahepatic accumulation of fats [6].

Risk factors, such as diabetes mellitus, age, sex, family history of hypertension and smoking are linked to hyperlipidemia [7], in addition to obesity and insulin resistance [6] [8]. According to Ghebreyesus, 2019 [9], lipidaemia, especially hypercholesterolemia, increases the risk of heart diseases and stroke, leading to about one-third of ischaemic heart diseases globally and resulting to approximately 29.7 million disabilities and 2.6 million deaths. Furthermore, these metabolic disorders present other complications like oxidative stress and inflammation [5]. The situation has become a global concern, with increasing prevalence over the years among all age groups. Findings reveal that approximately 17 million children and adolescents globally are overweight and prone to develop both hyperlipidemia and its consequences, while the annual incidence is 0.5% - 1.0% [10].

Liver diseases account for 7.9% of adult medical admissions in Nigeria [11], with liver cirrhosis and liver cancer accounting for almost two thirds of liv-

er-related diseases and hepatitis B virus infection being about 60% of chronic liver disease [12].

There is a relationship between chronic dysfunctional liver metabolism, such as liver cirrhosis and lipid profile [3]; thus, this study examined the changes in the liver morphology using ultrasonographic approach to ascertain its relationship with lipidaemia, while the main concern was to elucidate the most prevalent lipid profile parameter when ultrasonography is employed and there is a relationship between the liver span and lipid profile by assaying total cholesterol (TC), triglyceride (TG), low density lipoprotein (LDL) and high density lipoprotein (HDL), which are observed as the dominant proponents for the etiology of cardiovascular disorders. While increased TG is generally associated with fatty liver [2], it presents more as coronary heart disease in the elderly [13].

Histopathology is the primary diagnostic technique employed to detect defects in the architecture of the liver. However, this is usually invasive and could be associated with severe complications [14]. This elicited the desire to develop other techniques, non-invasive or less invasive, and have less or no complications, such as ultrasonography. Investigation of insulin concentration is another diagnostic protocol. This arose from the understanding that insulin does not only affect glucose metabolism, but also that of lipids in the peripheral circulation [15] [16]. Ultrasonography is considerably accurate and easy to perform, but also possess the disadvantage of being mostly qualitative, rather than quantitative, which should indicate the extent of the abnormality or deviation from normalcy [8] [17].

2. Methodology

This study adopted a prospective study that employed the biophysical method, undertaken in a Nigerian population of 30 subjects using the Taro-Yamane formula for sample size determination, which is mathematically illustrated as: $n = N/\{1 + N(e)2\}$, where; n = is sample size, N = is population under study and e = margin of error (0.05). Employing the 7.9% prevalence reported by Nwokediuko *et al.* 2013 [11], we obtain;

$$n = 652/\{1+652(0.05)\times 2\}$$

$$n = 652/\{1+32.6\times 2\}$$

$$n = 652/\{1+65.2\}$$

$$n = 652/66.2$$

Sample size = 9.8; approximately 10 research subjects. This was multiplied by 3 to make the sample size reasonable. This gives 30 as the sample size. Bio-data such as age, sex, occupation and state of origin was obtained through the use of request form at the centre. Liver parameters were assessed using an ultrasound scanning machine (SS 5 ultrasound scanner model by Sonostar Technologies Limited), using a convex probe 3.5 MHz. Also, phlebotomy was performed by collecting blood from subjects on fast for a period of about 10 hours and dis-

pensing same into a heparin container for analysis using the Semi-automated Chemistry analyzer (model Horron RD-171). The parameters assayed for are lipid profile, such as, Total Cholesterol, Triglyceride, High density Lipoprotein (HDL) Cholesterol and Low density Lipoprotein (LDL) Cholesterol.

Obtained data was analyzed using Statistical Programme for Social Sciences (SPSS) version 20 and analysis was done using ANOVA to compare the means of the parameters of the continuous data among groups and the least significance difference (LSD) was obtained for multiple comparison. P-value less than or equal to 0.05 was considered statistically significant, however Chi-square test was used to compare the categorical data between groups and all results presented as percentages and Pearson's correlation coefficients for bivariate correlation. Similarly, Ethical approval was obtained from the Research and Ethics Committee of the University of Port Harcourt and a verbal consent from the participants, but, their names were kept anonymous.

3. Results

Table 1 below shows that respondents are mainly aged 30 - 39 years, 12 (40.00%), with a female preponderance, 20 (66.67%) and obese, 16 (53.33%). There was Christian dominance, 25 (83.33%).

The lipid profile in Table 2 below shows that total cholesterol was mostly high, 18 (60.00%), mostly normal for triglyceride, 24 (80.00%), mostly low for high density lipoprotein cholesterol, 22 (73.33%) and mostly high for low density lipoprotein cholesterol, 14 (46.67%).

Table 3 below presents the association between maximum liver span and lipid profile. The maximum liver span was statistically significant to the concentration of triglyceride p-value (0.001) but not for total cholesterol p-value (0.084), high density lipoprotein cholesterol p-value (0.477) and low density lipoprotein cholesterol p-value (0.317).

Liver span, as depicted by the scatter plot in Figure 1 below shows a negative correlation with an increase in total cholesterol. This is shown with the proportionate decrease in the plasma lipid (total cholesterol) as the liver span increases. A regression equation is shown above, which can be used to calculate a parameter if one of the variables such as the liver span or total cholesterol value is known.

The scatter plot in Figure 2 below shows that liver span does not have a direct correlation with the concentration of plasma lipids.

Liver span, as depicted by the scatter plot in Figure 3 below shows a negative correlation with an increase in high density lipoprotein cholesterol. This is shown with the proportionate decrease in the plasma lipid (high density lipoprotein cholesterol) as the liver span increases. A regression equation is shown above, which can be used to calculate a parameter if one of the variables such as the liver span or the high density lipoprotein cholesterol value is known.

Like Total CHO and HDLc, LDLc in Figure 4 also shows a negative correlation that is proportionate with the span of the liver.

Table 1. Socio-demographic characteristics.

Variable	Frequency	Percent
Age (years)		
30 - 39	12	40.00
40 - 49	9	30.00
50 - 59	5	16.67
60 - 69	4	13.33
Sex		
Female	20	66.67
Male	10	33.33
Body mass index (BMI) (kg/m²)		
Underweight (≤18.5)	-	-
Normal weight (18.5 - 24.9)	7	23.33
Overweight (25 - 29.9)	7	23.33
Obese (≥30)	16	53.33
Religion		
Muslim	3	10.00
Christian	25	83.33
Others (specify)	2	6.67

Table 2. Lipid profile of respondents.

Variable	Frequency	Percent
Total cholesterol (2.5 - 5.12)		
Low	3	10.00
Normal	9	30.00
High	18	60.00
Triglyceride (0.3 - 2.0)		
Low	-	-
Normal	24	80.00
High	6	20.00
HDLc (1.04 - 1.4)		
Low	22	73.33
Normal	4	13.33
High	4	13.33
LDLc (2.6 - 3.3)		
Low	13	43.33
Normal	3	10.00
High	14	46.67

*HDLc = High density lipoprotein cholesterol, LDLc = Low density lipoprotein cholesterol.

Table 3. Correlation between maximum liver span to lipid profile.

Maximum liver span	Chi-square	Degree of freedom
Total cholesterol	0.084	9
Triglyceride	0.001	9
High density lipoprotein cholesterol	0.477	9
Low density lipoprotein cholesterol	0.317	9

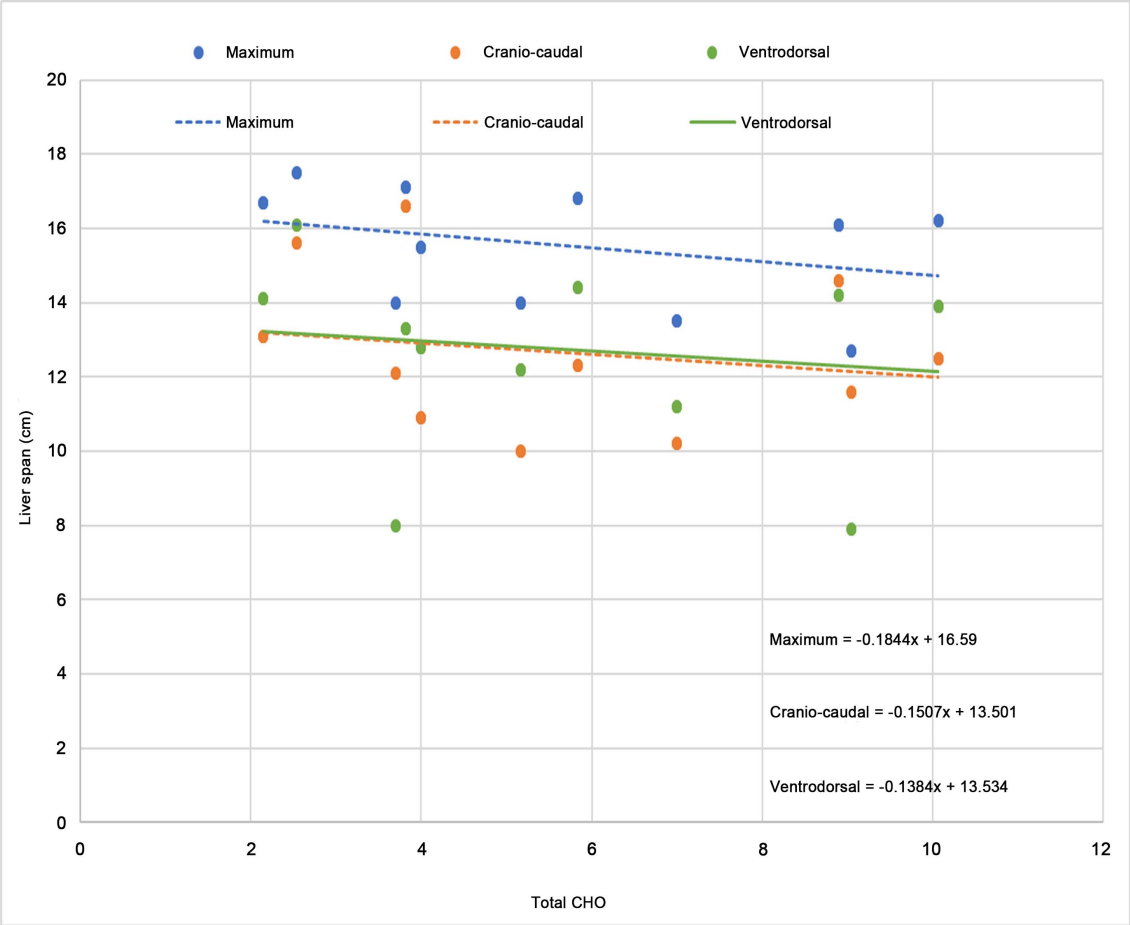

Figure 1. Scatter plot of Liver span (Maximum, Craniocaudal and Ventro-dorsal) and Total CHO. *Total CHO = total cholesterol.

4. Discussion of Findings

Majority of the respondents were aged 30 - 39 years, 12 (40.00%), with female preponderance, 20 (66.67%) and majority obese, 16 (53.33%). The majority obesity in this study is in tandem with (WHO, 2011) [18], that obesity is among the metabolic disorders associated with hyperlipidemia and is attributed to the morbidity and mortality linked with hyperlipidemia and its prevalence has doubled in the last 3 decades. The finding also agrees with those of Katsiki *et al.* 2016; Bae *et al.* 2010 [6] [8] which posited that both obesity and insulin resistance are prominent risk factors for developing hyperlipidemia. The obese majority is not

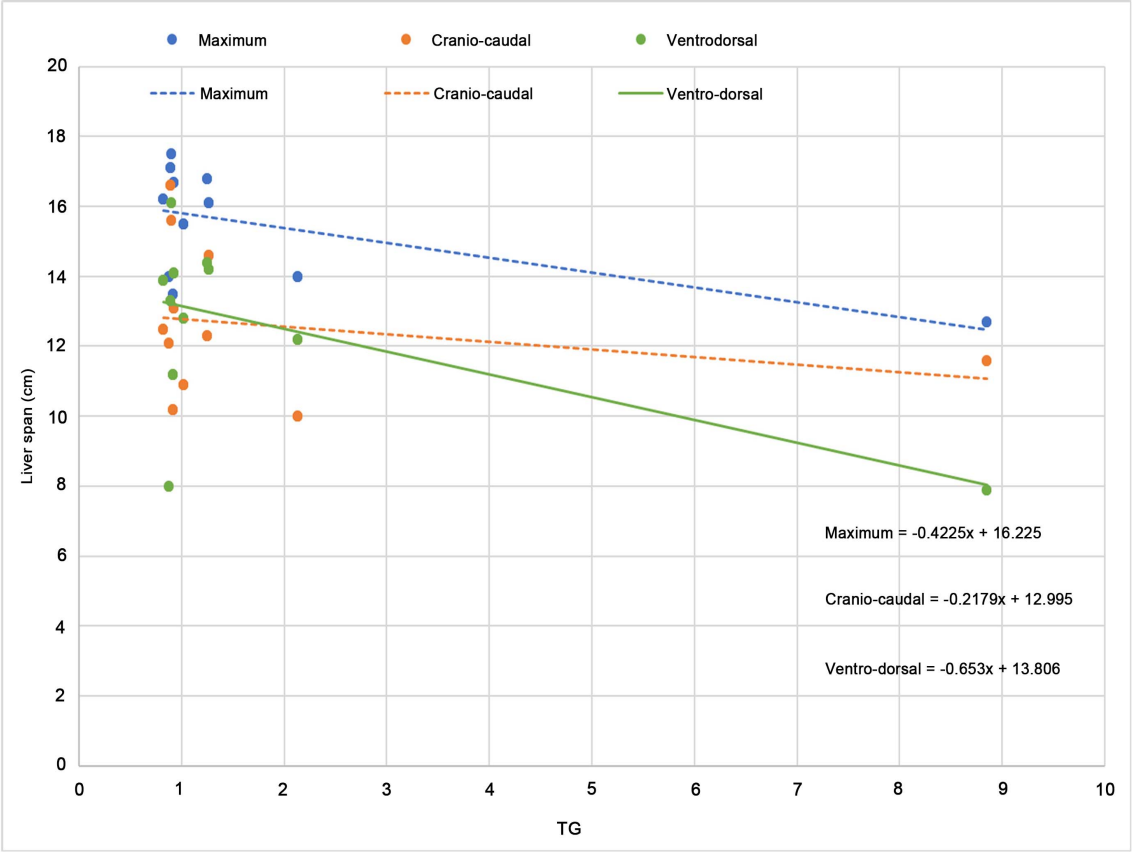

Figure 2. Scatterplot of Liver span (Maximum, Craniocaudal and Ventro-dorsal) and TG. *TG = triglyceride.

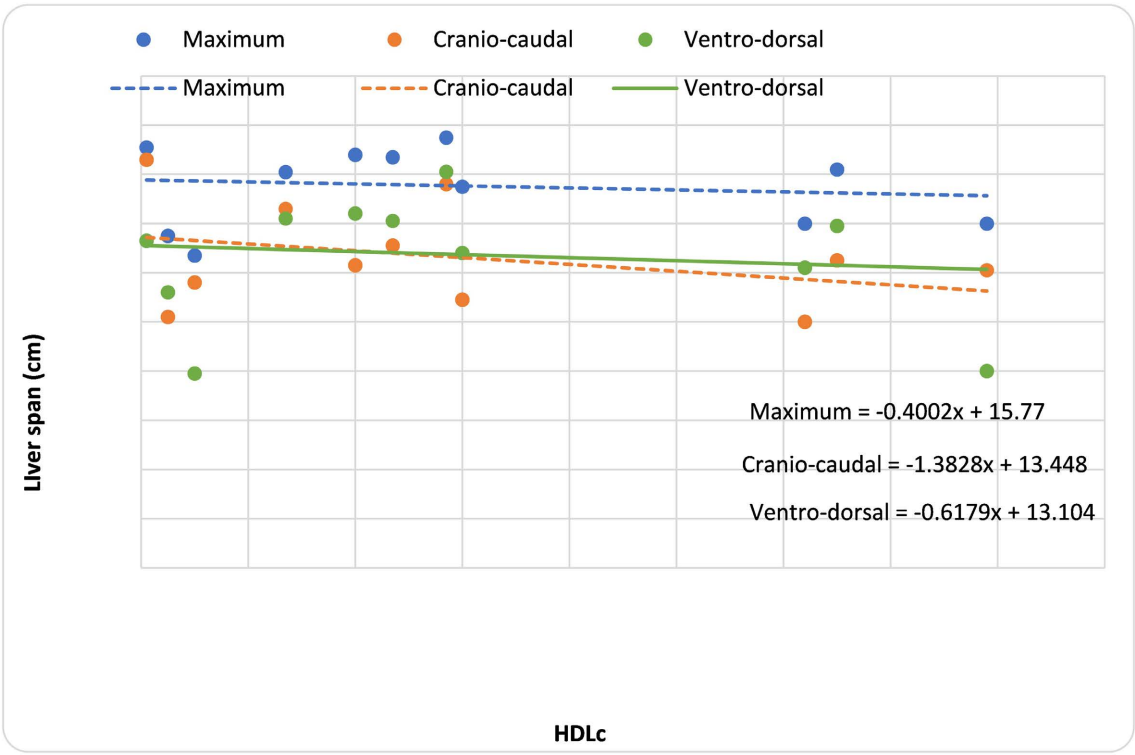

Figure 3. Scatter plot of HDLc to maximum liver span. *HDLc = high density lipoprotein cholesterol.

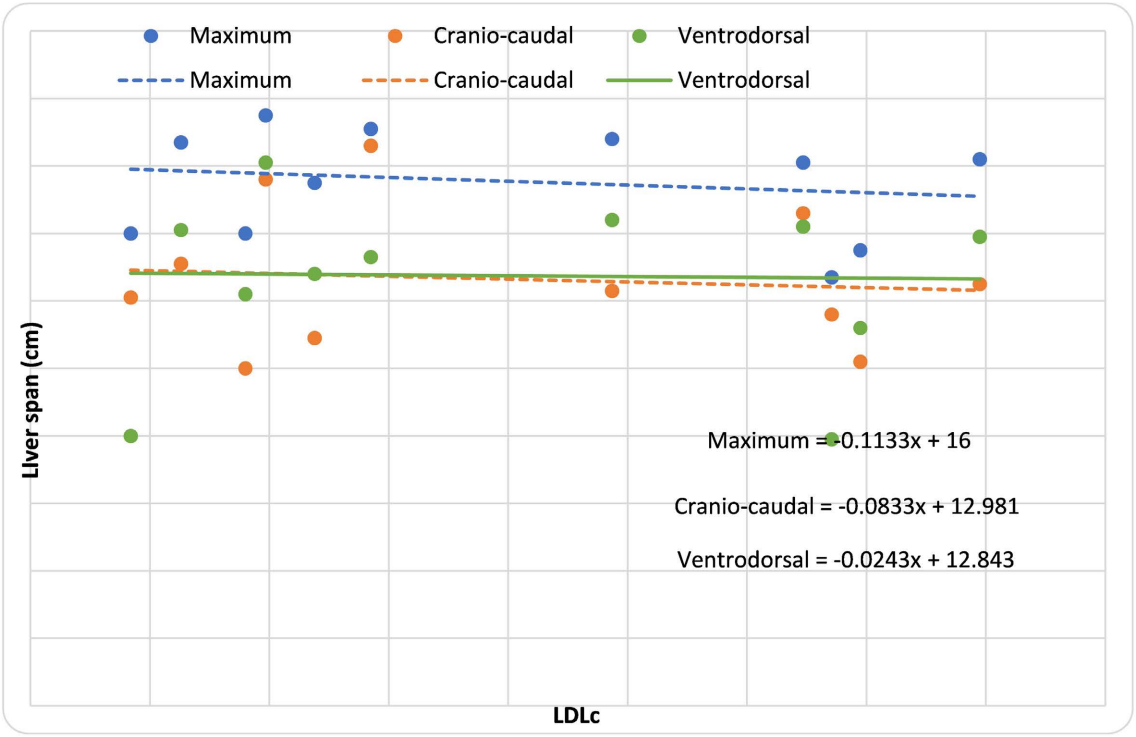

Figure 4. Scatter plot of LDLc to maximum liver span. LDLc = low density lipoprotein cholesterol.

due solely to lifestyle, as some could be inherent. Obesity has usually been associated with lifestyle consequences and a risk factor of the pathogenesis of hyperlipidemia. This observation agreed with that of Rao and Khan 2017 [19] who conceptualized the close association between obesity and hyperlipidemia. Lifestyle conditions such as the chronic consumption of alcohol have been linked to increased plasma lipids (hyperlipidemia), with virtually all plasma lipids (triglycerides, total cholesterol, HDLc and LDLc) according to Bhusal *et al.* 2017 [20]. Also, contrary to our finding were individuals aged 30 - 39 years were more prone to hyperlipidemia, [21], reported that the fifth decade of life were more prone to the disease condition. Obesity is also associated with both hyperglycemia and this relationship is confirmed by Jain *et al.* 2014 [22], Al-Jameil *et al.* 2014 [23] and Belay *et al.* 2014 [24].

This study observed that total cholesterol was the highest, 18 (60.00%), mostly normal for triglyceride, 24 (80.00%), with none being low, while it was mostly low for high density lipoprotein cholesterol, 22 (73.33%) and mostly high for low density lipoprotein cholesterol, 14 (46.67%) and followed by low, 13 (43.33%) and the least was normal, 3 (10.00%). The finding of HDLc being mostly low, contradicts the observation of Changchien *et al.* 2013 [7] who reported increase in lipid profile in metabolic abnormalities and cardiovascular diseases are mostly associated with increase in HDLc, rather than the other plasma lipids. This difference may be due to difference in study settings, as well as, the predominating class of metabolic disorders of the respondents in the respective studies. The finding of triglyceride being mostly normal in this study is similar to that re-

ported by Liu *et al.* 1990 [25], that was conducted more than three decades ago. Its relevance up till date may indicate that is the normal nature of the lipid in hepatic abnormalities. However, it can be due to similarity in the two study populations. The finding of LDLc having a statistically significant relationship with hyperlipidemia is in tandem with the observation of Lee, 2015 [26], and colleagues, with the similarity thought mainly associated with the peculiarity of food consumed by the respondents in the two studies, as well as, possible similarities in setting. Other than these, one may consider mere co-incidence. The observation of normal triglycerides is contradictory to the findings of Lee *et al.* 2012 [27], Parekh and Anania 2007 [28] and McCrindle 2006 [29] which reported reduced serum concentrations of triglycerides and total cholesterol in metabolic abnormalities of the liver.

Maximum liver span was statistically significant to the concentration of triglyceride p-value (0.001) but was not statistically significant for total cholesterol p-value (0.084), high density lipoprotein cholesterol p-value (0.477) and low density lipoprotein cholesterol p-value (0.317). Chatrath *et al.* 2012 [30] and Targher *et al.* 2010 [31] had similar findings for total triglycerides, but varied for total cholesterol, HDLc and LDLc. The difference in some of the findings may be due to variation in socio-cultural and lifestyle conditions, such as, exercise or food, and the prevailing environmental circumstances among the two study populations.

Another researcher had reported that a relationship between obesity and hyperlipidaemia with female preponderance. This study also demonstrated obvious female preponderance. The precise role of hypertriglyceridemia in atherogenesis is little known among women and older patients, while the role of triglyceride in CVDs risk is controversial.

5. Summary

This study was conducted in the University of Port Harcourt Teaching Hospital, while the sample analysis was done in HMG Hospital Ltd within Rivers State. The respondents were mainly aged 30 - 39 years, 12 (40.00%), mainly females, 20 (66.67%) and obese, 16 (53.33%). They were also mainly Christians, 25 (83.33%).

A random sampling technique was employed to select the respondents, while the Taro-Yamene formula was used to calculate the sample size and data was analyzed with SPSS version 20. The total cholesterol was the highest, 18 (60.00%), normal for triglyceride, 24 (80.00%), low for high density lipoprotein cholesterol, 22 (73.33%) and high for low density lipoprotein cholesterol, 14 (46.67%). Maximum liver span was statistically significant to triglyceride concentration p= (0.001) but not for total cholesterol p = (0.084), high density lipoprotein cholesterol p = (0.477) and low density lipoprotein cholesterol p = (0.317). In conclusion, liver span is a predictive tool for the diagnosis of dyslipidaemia. Also a regression equation was propounded which can be used to predict the liver span using the lipid values and vis-versa.

Conflicts of Interest

The author declares no conflicts of interest regarding the publication of this paper.

References

[1] Lai, S.W., Tan, C.K. and Ng, K.C. (2002) Epidemiology of Fatty Liver in a Hospital-Based Study in Taiwan. *Southern Medical Journal*, **95**, 1288-1292. https://doi.org/10.1097/00007611-200295110-00012

[2] Wang, L.Y., Chen, J.F. and Lin, M.P. (2003) A Study on the Incidence and Associated Factors of Fatty Liver from a Physical Examination Center. *Nursing Journal*, **50**, 57-64.

[3] Ghadir, M.R., Riahin, A.A., Avazpour, A., Nooranipour, M. and Habibinejad, A.A. (2011) The Relationship between Lipid Profile and Severity of Liver Damage in Cirrhotic Patients. *Hepatitis Monthly*, **10**, 285-288.

[4] Sriram, B.M. (2013) SRB Manual of Surgery. Jaypee Brothers Medical Publishers Limited, New Delhi.

[5] Rajeswari, S., Arun, K., Ghandi, M. and Swaminathan, S. (2014) Association between Lipid Profile and Liver Function Tests in Diabetic Patients. *International Journal of Pure and Applied Bioscience*, **2**, 26-31.

[6] Katsiki, N., Mikhailidis, D.P. and Mantzoros, C.S. (2016) Non-Alcoholic Fatty Liver Disease and Dyslipidemia: An Update. *Metabolism*, **65**, 1109-1123. https://doi.org/10.1016/j.metabol.2016.05.003

[7] Changchien, C., Wang, J., Tsai, T. and Hung, C. (2003) Correlation between Fatty Liver and Lipidemia in Taiwanese. *Journal of Medical Ultrasound*, **11**, 60-65. https://doi.org/10.1016/S0929-6441(09)60043-6

[8] Bae, J.C., Cho, Y.K., Lee, W.Y., Seo, H.I., Rhee, E.J. and Park, S.E. (2010) Impact of Nonalcoholic Fatty Liver Disease on Insulin Resistance in Relation to HbA1c Levels in Non-Diabetic Subjects. *American Journal of Gastroenterology*, **105**, 2389-2395. https://doi.org/10.1038/ajg.2010.275

[9] Ghebreyesus, T.A. (2019) Global Health Observatory Data. World Health Organization.

[10] Lobstein, T., Baur, L. and Uauy, R. (2004) IASO International Obesity Task Force. Obesity in Children and Young People: A Crisis in Public Health. *Obesity Review*, **5**, 4-85. https://doi.org/10.1111/j.1467-789X.2004.00133.x

[11] Nwokediuko, S.C., Osuala, P.C., Uduma, U.V., Alaneme, A.K., Onwuka, C.C. and Mesigo, C. (2013) Pattern of Liver Disease Admissions in a Nigerian Tertiary Hospital. *Nigerian Journal of Clinical Practice*, **16**, 339-342. https://doi.org/10.4103/1119-3077.113458

[12] Nwokediuko, S., Ijoma, U.N. and Obienu, O. (2011) Liver Cancer in Enugu, Southeast Nigeria. *Insight Bioinformatics*, **1**, 1-5. https://doi.org/10.5567/BIOINFO-IK.2011.1.5

[13] Akahoshi, M., Amasaki, Y. and Soda, M. (2001) Correlation between Fatty Liver and Coronary Risk Factors: Apopulation Study of Elderly Men and Women in Nagasaki, Japan. *Hypertension Resource*, **24**, 337-343. https://doi.org/10.1291/hypres.24.337

[14] Limanond, P., Raman, S.S., Lassman, C., Sayre, J., Ghobrial, R.M. and Busuttil, R.W. (2004) Macrovesicular Hepatic Steatosis in Living Related Liver Donors: Correlation

between Computed Tomography and Histologic Findings. *Radiology*, **230**, 276-280. https://doi.org/10.1148/radiol.2301021176

[15] Bjornstad, P. and Eckel, R.H. (2018) Pathogenesis of Lipid Disorders in Insulin Resistance: A Brief Review. *Curriculum in Diabetes and Reproduction*, **18**, Article No. 127. https://doi.org/10.1007/s11892-018-1101-6

[16] Taskinen, M.R., Adiels, M., Westerbacka, J., Soderlund, S., Kahri, J. and Lundbom, N. (2011) Dual Metabolic Defects Are Required to Produce Hypertriglyceridemia in Obese Subjects. *Arteriosclerosis, Thrombosis and Vascular Biology*, **31**, 2144-2150. https://doi.org/10.1161/ATVBAHA.111.224808

[17] Kodama, Y., Ng, C.S., Wu, T.T., Ayers, G.D., Curley, S.A. and Abdalla, E.K. (2007) Comparison of CT Methods for Determining the Fat Content of the Liver. *American Journal of Roentgenology*, **188**, 1307-1312. https://doi.org/10.2214/AJR.06.0992

[18] World Health Organization (2011) Global Status Report on Non-Communicable Diseases 2010. World Health Organization, 22-31.

[19] Rao, B.R. and Khan, S.A. (2017) Study of Lipid Profile in Non-Alcoholic Fatty Liver Disease. *Journal of Dental and Medical Sciences*, **16**, 46-49.

[20] Bhusal, K.R., Simkhada, R. and Nepal, P. (2017) Lipid Profile in Different Grades of Ultrasonic Non-Alcoholic Fatty Liver Disease. *Journal of the College of Medical Science*, **13**, 258-261. https://doi.org/10.3126/jcmsn.v13i2.17773

[21] Khalil, U.P., Paudel, B., Gurung, G., Hu, Y.S. and Kuo, C.W. (2019) Correlational Study of Nonalcoholic Fatty Liver Disease Diagnosed by Ultrasonography with Lipid Pro le and Body Mass Index in Adult Nepalese Population. *Journal of Medical Ultrasound*, **27**, 19-25. https://doi.org/10.4103/JMU.JMU_53_18

[22] Jain, H.R., Shetty, V., Singh, G.S. and Shetty, S. (2016) A Study of Lipid Profile in Diabetes Mellitus. *International Journal of Scientific Studies*, **4**, 56-61.

[23] Al-Jameil, N., Khan, F.A., Arjumand, S., Khan, M.F. and Tabassum, H. (2014) Associated Liver Enzymes with Hyperlipidemic Profile in Typediabetes Patients. *International Journal of Clinical and Experimental Pathology*, **7**, 4345-4349.

[24] Belay, Z., Daniel, S., Tedla, K. and Gnanasekaran, N. (2014) Impairment of Liver Function Tests and Lipid Profiles in Type 2 Diabetic Patients Treated at the Diabetic Center in Tikur Anbessa Specialized Teaching Hospital (Tasth), Addis Ababa, Ethiopia. *Journal of Diabetes and Metabolism*, **5**, Article ID: 1000454. https://doi.org/10.4172/2155-6156.1000454

[25] Liu, C., Zhou, W.N., Lü, Z., Wang, X.T. and Qiu, Z.H. (2018) The Associations between Liver Enzymes and the Risk of Metabolic Syndrome in the Elderly. *Experimental Gerontology*, **106**, 132-136. https://doi.org/10.1016/j.exger.2018.02.026

[26] Lee, S.H., Kim, D., Baek, M.Y., Tchah, H., Kim, Y.S., Ryoo, E. and Kim, Y.M. (2015) Abdominal Subcutaneous Fat Thickness Measured by Ultrasonography Correlates with Hyperlipidemiaand Steatohepatitis in Obese Children. *Pediatric Gastroenterology, Hepatology and Nutrition*, **18**, 108-114. https://doi.org/10.5223/pghn.2015.18.2.108

[27] Lee, H.H., Choi, S.K. and Seo, J.S. (2012) Obesity Index and Related Factors among Elementary School Students Visiting Pediatric Department of General Hospital. *Journal of Korean Dietetics Association*, **18**, 186-199. https://doi.org/10.14373/JKDA.2012.18.2.186

[28] Parekh, S. and Anania, F.A. (2007) Abnormal Lipid and Glucose Metabolism in Obesity: Implications for Nonalcoholic Fatty Liver Disease. *Gastroenterology*, **132**, 2191-2207. https://doi.org/10.1053/j.gastro.2007.03.055

[29] McCrindle, B.W. (2006) Hyperlipidemia in Children. *Thrombosis Research*, **118**, 49-58. https://doi.org/10.1016/j.thromres.2005.01.006

[30] Chatrath, H., Vuppalanchi, R. and Chalasani, N. (2012) Dyslipidemia in Patients with Nonalcoholic Fatty Liver Disease. *Seminars in Liver Disease*, **32**, 22-29. https://doi.org/10.1055/s-0032-1306423

[31] Targher, G., Day, C.P. and Bonora, E. (2010) Risk of Cardiovascular Disease in Patients with Nonalcoholic Fatty Liver Disease. *New England Journal of Medicine*, **363**, 1341-1350. https://doi.org/10.1056/NEJMra0912063