

ORIGINAL ARTICLE

OPEN ACCESS

Remnant Cholesterol and Leptin: Any Association in Normal Weight, Over Weight and Obese Individuals in South East Nigeria

Chinonso J Nnamdi¹   ▪ Amah K Ubuo¹   ▪ Anaelechi J Onuegbu¹   ▪ Madu J Olisekodiaka²  
Chima H Okpara³   ▪ Ekenedirichukwu J Okwara¹   ▪ Adamma R Analike¹   ▪ Chidiadi M Njoku¹  

¹ Department of Chemical Pathology, Faculty of Basic Clinical Sciences, Nnamdi Azikiwe University, Awka, Nigeria

² Clinical Chemistry, Biomedical Sciences, Health Sciences, Ladoke Akintola University of Technology, Ogbomoso, Nigeria

³ Department of Chemical Pathology, University of Calabar, University of Calabar Teaching Hospital, Calabar, Nigeria

Introduction: Leptin circulating levels increase with weight gain and decrease with weight loss. The amount of leptin circulating in the body is proportional to the amount of fat of an individual. We investigated the association of leptin and remnant cholesterol in normal weight, overweight and obese participants in south east Nigeria.

Materials and Methods: This is a cross-sectional random study. A total of 90 apparently healthy obese, overweight and normal weight participants who met the inclusion criteria were randomly enrolled into the study. They were grouped using their body mass index of 18.5 to 24.9 kg/m² (normal weight), 25 to 29.9 kg/m² (overweight) and ≥30 kg/m² (obese). The measured parameters were analyzed using standard methods.

Results: Our results showed that there was progressive increase of remnant cholesterol among the group but there was no statistical difference ($p > 0.05$) in the mean calculated remnant cholesterol (mmol/l) between the obese (0.72 ± 0.4 mmol/l), overweight (0.68 ± 0.46), and normal weight (0.50 ± 0.28 mmol/l) participants. There was significant difference in the mean level of Leptin (ug/l) of the obese participants compared to normal weight participants (0.63 ± 0.29 vs 0.25 ± 0.26 , 0.001) and overweight when compared to normal weight (0.51 ± 0.27 vs 0.25 ± 0.26 , 0.003). Remnant cholesterol did not correlate with body mass index (BMI) in those that are obese, overweight or normal weight. Leptin correlated positively with BMI but inversely with Remnant cholesterol.

Conclusion: The result obtained from this study suggests that leptin has a positive relationship with BMI and calculated Remnant cholesterol does not appear to have a direct relationship with obesity. There was no association between leptin and remnant cholesterol among the groups.

Keywords: Leptin, remnant cholesterol, obesity, BMI, cardiovascular disease, lipid profile

Introduction

Obesity is one of the leading preventable causes of death worldwide. (1). The World Health Organizations (WHO) technical report series of 1995 indicated that body weight significantly impacts an individual's health and disease status (2). Bakari et al., in 2007, reported these abnormalities of body weight to be

expected in two suburban communities in Northern Nigeria at the rates of 13.1% for obesity, 18.5% for overweight and 6.7% for underweight (3). Data on obesity have been commonly reported in the past among specific risk groups, such as hypertensive and diabetic populations, many of which are hospital-based studies (4, 5). Other community-based studies

Corresponding Author: Chinonso J Nnamdi; Department of Chemical Pathology, Faculty of Basic Clinical Sciences, Nnamdi Azikiwe University, Awka, Nigeria
E-mail: cj.nnamdi@unizik.edu.ng
ORCID: 0000-0003-3795-1275

Received: Aug 13, 2022 **Accepted:** Oct 27, 2022
Published: Dec 9, 2022

This is an Open Access article distributed under the terms of Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, or reproduction in any area, original work is properly cited.

The Ulutas Medical Journal © 2022



that are now emerging have been reported among rural or semi-urban dwellers (6).

Obesity, defined by the World Health Organization as a Body Mass Index (BMI) ≥ 30 kg/m², has become an epidemic dimension in the world, with Nigeria also having its share of the burden, as reported by Akarolo-Anthony et al.(7). This current trend of obesity has been attributed to the socio-economic and epidemiological transition of Nigeria from a lower income country to a middle-income country, resulting in the change of lifestyle behaviors and dietary intake. As a result, many Nigerians now live sedentary lifestyles while consuming a diet with excess calories, as observed by Iloh et al (8). Lopez et al.(9), reported CVD in developing countries causes twice as many deaths as HIV, malaria, and tuberculosis combined. According to earlier speculation, almost all unexpected deaths of medical origin in Nigeria are due to cardio-vascular causes. The association among dyslipidemia, obesity and hypertension is well established (10), and all are major risk factors for the development of CVD, a leading cause of visits to physicians and cause of death. Dyslipidemia is becoming increasingly common in Africa.

Some studies (11, 12) claim that leptin serum concentrations in healthy individuals positively correlate with body fat content but correlate negatively when energy intake is reduced, and energy stores in fat are declining. Leptin levels are correlated positively with several endocrine substances, such as insulin, glucocorticoid, thyroids, and testosterone, as observed by Chu et al.(13). Similar studies also indicate that it may mediate some endocrine-related processes, like the onset of puberty (14). Chu et al.(13), suggested that impaired or modified receptor function may be associated with certain medical conditions other than obesity, such as cardiovascular disease and certain types of cancer. Research findings also indicate that

leptin can act as a growth factor in the fetus and the neonate. It has been reported that obesity is a significant risk factor for the development of diabetes mellitus (15).

Remnant cholesterol is primarily chylomicron and VLDL, from which most triglyceride has been removed, such that each remnant particle contains about 40 times more cholesterol than LDL (16). According to a study, high remnant cholesterol is more predictive of myocardial infarction than any other lipid particle (17). Remnant cholesterol is significantly predictive of coronary artery disease in patients with normal total cholesterol (18,17).

Varbo et al. (19) suggested that remnant cholesterol was a mediator from obesity to ischemic heart disease; part of the higher risk of ischemic heart disease seen in obese individuals was explained by higher remnant cholesterol concentrations. However, it is currently unknown whether there is an association between high concentrations of remnant cholesterol as a mediator between obesity and a high risk of developing cardiovascular disease and leptin as a mediator in the development of obesity found in average weight, overweight, and obese individuals alike.

Materials and Methods

This is a cross-sectional study designed to compare the serum level of Leptin, some lipid fractions, and Remnant Cholesterol in normal, overweight, and obese participants. Based on the calculated sample size, 90 consenting participants that fulfilled the inclusion criteria were randomly recruited for the study. Informed consent from the participants was sought and obtained before enrollment into the study. Ethical approval was obtained from the Ethics Committee (NAUTH/CS/66/VOL.11/154/2018/088) of Nnamdi Azikiwe University Teaching Hospital Nnewi, and informed consent was sought from participants. Questionnaires were administered and served as a primary instrument for this study. The

questionnaires were structured to reflect the health issues relevant to the study's objectives; the study comprised 90 obese and non-obese subjects, who were picked by non-probability sampling. Among them, 30 participants were known obese, 60 participants were non-obese, and between 20-50 years. The participants were labeled Normal weight (BMI 18.5-24.9 kg/m²), over-weight (BMI 25-29.9kg/m²) Obese (≥ 30 kg/m²). BMI was measured as weight in kilogram 0.01 divided by height squared (m²), WC was measured with a tape to the nearest 0.1cm at the end of a normal expiration at the midpoint between the subcostal plane and the iliac crest of an exposed abdomen. HC was measured at the largest standing horizontal circumference of the buttock to determine the waist-to-hip ratio (WHR=WC/HC). Hypertension was defined as systolic blood pressure ≥ 140 mmHg, and diastolic blood pressure ≥ 90 mmHg. A fasting blood sample was obtained in the morning between 08:00-09:00 h, about 7ml, through venipuncture from each participant. Aliquots were drawn into a fluoride oxalate container for plasma glucose estimation, and later on, a non-fasting blood sample was obtained from the participants, and aliquots were drawn into plain tubes and allowed to clot at room temperature.

The quantitative determination of serum leptin was performed using the sandwich Enzyme-Linked Immunosorbent Assay (ELISA). Technique, with commercially available reagent kit MONOCENT. Leptin (sandwich) ELISA, (CAT no: EL9-1059) (LOT no: 200610) by Monocent USA. The fasting lipid profile was assessed using commercially available kits (Randox). Serum total cholesterol and high-density lipoprotein HDLc were determined by the cholesterol oxidase method by Allain et al. (20) and serum triglyceride by glycerol kinase Trinder (21), and LDLc was calculated using the Friedwald formula(22). Furthermore, Remnant Cholesterol was calculated using the formula. $Rc=Tc-HDLc-LDLc$ by Dave-Feldman (2017). Fasting plasma glucose was determined using the enzymatic

colorimetric method of Trinder (21). Statistical analysis was performed to compare the two groups using ANOVA. The values were expressed as the Mean $\pm$ S.D. Values were regarded as significant if p was ≤ 0.05 . Pearson correlation coefficient was used to correlate the parameters estimated.

Results

Table-1 shows the mean anthropometric markers of all participants. There was no significant difference in the diastolic and systolic blood pressures. There was a significant increase in the body mass index of all the participants ($p<0.001$). There was a significant difference in the participants' waist-to-hip ratio. Table 2; shows the mean of the various fasting biochemical parameters of obese, overweight, and normal weight groups. A significant change was observed in the fasting plasma Triglyceride (TG) and very low-density lipoprotein (VLDL). ($p<0.001$) in the obese subjects when compared with the corresponding control values. No significant change was observed in other lipid parameters. Table 3; show the mean of the various non-fasting biochemical parameters of obese, overweight, and normal weight group. A significant change was observed in the non-fasting plasma Triglyceride (TG) ($p<0.004$) in the obese subjects when compared with the corresponding control values. No significant change was observed in other lipid parameters. Table 4; Show the mean serum leptin, remnant cholesterol, and fasting blood sugar in the obese, overweight, and normal-weight participants. There was a significant difference in the serum leptin level between the normal-weight participants (0.25 ± 0.26) and overweight participants (0.51 ± 0.27) at ($P<0.003$). There was a significant difference between the normal weight (0.25 ± 0.26) and obese participants (0.63 ± 0.29 ; $p<0.001$). There was no difference in the mean RC between the obese and overweight compared to the control. There was statistical significance in the fasting blood sugar

Table 1. Anthropometric maker and indices of obesity. It highlights the BMI, WHR, diastolic and systolic blood pressures

GROUPS	SBP (mmHg)	DBP (mmHg)	BMI (Kg/m ²)	WHR
NORMAL WEIGHT (A)	115.28±11.89	75.59±10.03	21.75±1.82	0.85±0.05
OVER WEIGHT (B)	118.92±25.02	78.4±9.55	27.11±1.02	0.86±0.07
OBESE (C)	127±10.86	85.64±12.25	33.85±4.51	0.96±0.1
f-test	3.310	6.246	123.387	18.038
p-value	0.042*	0.003*	<0.001*	<0.001*
A vs B	1.000	1.000	<0.001*	1.000
A vs C	0.040*	0.003*	<0.001*	<0.001*
B vs C	0.287	0.056	<0.001*	<0.001*

Table 2. Levels of fasting HDL-C, LDL-C, VLDL-C, TG, and TC in normal weight, over weight and obese groups

GROUPS	F-HDL-C (mmol/L)	F-LDL-C (mmol/L)	F-VLDL-C (mmol/L)	F-TG (mmol/L)	F-TC (mmol/L)
NORMAL WEIGHT (A)	1±0.21	1.77±0.67	0.47±0.22	1.09±0.51	3.39±0.66
OVER WEIGHT (B)	0.95±0.09	1.52±0.34	0.76±0.31	1.72±0.6	3.47±0.42
OBESE (C)	1±0.17	1.84±0.38	0.82±0.23	1.85±0.59	3.45±0.53
f-test	0.816	3.006	9.391	8.638	0.135
p-value	0.446	0.055	<0.001*	<0.001*	0.874
A vs B	0.732	0.201	<0.001*	<0.001*	1.000
A vs C	1.000	1.000	<0.001*	<0.001*	1.000
B vs C	0.906	0.067	0.019*	0.030*	1.000

Table 3. Levels of non-fasting HDL-C, LDL-C, VLDL-C, TG and TC in normal weight, over weight and obese groups

GROUPS	NF-HDL-C (mmol/L)	NF-LDL-C (mmol/L)	NF-VLDL-C (mmol/L)	NF-TG (mmol/L)	NF-TC (mmol/L)
NORMAL WEIGHT (A)	1.05±0.27	1.87±0.68	0.44±0.23	1.02±0.52	3.42±0.8
OVER WEIGHT (B)	1.08±0.3	1.56±0.47	0.66±0.35	1.67±0.93	3.32±0.47
OBESE (C)	1.07±0.23	1.95±0.58	0.62±0.22	1.37±0.53	3.66±0.75
f-test	0.088	3.167	5.031	6.023	1.625
p-value	0.916	0.048*	0.009*	0.004*	0.204
A vs B	1.000	0.176	0.014*	0.003*	1.000
A vs C	1.000	1.000	0.049*	0.182	0.253
B vs C	1.000	0.058	1.000	0.418	0.614

Table 4. Levels of RC, and FBS in normal weight, over weight and obese groups

GROUPS	RC (mmol/l)	FBS (mmol/L)	Leptin (ug/l)
NORMAL WEIGHT (A)	0.5±0.28	3.46±0.79	0.25±0.26
OVER WEIGHT (B)	0.69±0.46	4.46±1.82	0.51±0.27
OBESE (C)	0.72±0.4	4.94±1.26	0.63±0.29
f-test	2.853	8.740	13.038
p-value	0.064	<0.001*	<0.001*
A vs B	0.199	0.021*	0.003*
A vs C	0.095	<0.001*	<0.001*
B vs C	1.000	0.647	0.337

Table 5. Correlations of LEPTIN and RC in normal weight, overweight and obese groups

PARAMETERS	R	P-VALUE
Leptin vs RC in normal weight group	-0.088	0.648
Leptin vs RC in over weight group	-0.081	0.701
Leptin vs RC in obese group	-0.107	0.611

* Significance at p<0.05; RC=Remnant Cholesterol

level between the normal weight participants (3.46 ± 0.79) and overweight (4.46 ± 1.82) at ($P < 0.021$). Also between the normal weight and obese participants (3.46 ± 0.79 vs. 4.94 ± 1.26) at ($P < 0.001$). No difference was observed in the level of the calculated remnant cholesterol among the groups. Table 5 shows the Pearson correlation of leptin association and remnant cholesterol in groups; there was no significant correlation of leptin with remnant cholesterol in the groups as shown. Normal weight ($r = -0.088$; $p < 0.648$), overweight ($r = -0.081$; $p < 0.701$) and obese ($r = -0.107$; $p < 0.611$).

Discussion

Leptin expression and secretion correlate with body fat and adipocyte, as Sone et al. (23) reported. Leptin reduces appetite and body weight. The paradoxical co-existence of obesity and hyperleptinemia suggests the pathology of leptin resistance. High levels of leptin were observed in obese participants ($0.63 \pm 0.29 \mu\text{g/l}$) than in average weight ($0.25 \pm 0.26 \mu\text{g/l}$; $p < 0.001$). There was an increased significant difference in the leptin level between the overweight ($0.51 \pm 0.27 \mu\text{g/l}$) participants when compared to the average weight ($0.25 \pm 0.26 \mu\text{g/l}$) participant at $p < 0.003$. Reports from different study populations have demonstrated that serum leptin concentrations are elevated in human obesity in proportion to the degree of adiposity. (24,25). This increase in leptin correlated with the BMI and WHR parameters in all the obese and overweight participants. This is also corroborated by the study conducted by Masoud et al. (26), who also reported a significantly high level of leptin in obese subjects. Considine et al. (11) observed 4 times increased serum leptin levels in obese subjects compared to non-obese subjects and a significant direct association of serum leptin with BMI. This has been related to the assertion that obesity may be partly due to leptin resistance. From our results, there is an increased level of remnant cholesterol level in

the groups in average weight (0.50 ± 0.28 mmol/l), overweight (0.69 ± 0.46 mmol/l), and obese (0.72 ± 0.4 mmol/l) participants above the projected reference range and percentile (0.48 mmol/l) by Varbo et al. (19), this shows that race might have a role in the pathophysiology of atherosclerosis induced by remnants cholesterol. There was no significant statistical difference in the level of remnant cholesterol among the groups. This shows the association of remnant cholesterol and cardiovascular disease is not caused only by adiposity, it can be explained by remnant cholesterol's ability to cause atherosclerosis in the arterial wall, which is a process that has been assumed to be driven by remnant cholesterol concentration in the bloodstream, independent of it a reason for elevations in overweight and obesity, this finding is in correspondence with Varbo et al., (27). The other lipid fractions, fasting TG and VLDL, were significantly elevated in obese participants when compared to corresponding groups (0.82 ± 0.23 mmol/L) obese to (0.47 ± 0.22 mmol /L) average weight at $p < 0.001$ for VLDL, and (1.85 ± 0.59 mmol/L) obese to (1.09 ± 0.51 mmol/L) average weight at $p < 0.001$ for TG. There is also a significant difference in the fasting blood glucose among the groups. There was no significant difference among this study's other lipid fractions.

Conclusion

From the study findings, there is a progressive increase in the level of calculated remnant cholesterol among the groups, but it was not statistically significant, and there was an increase in the level of leptin among the groups. There was no significant correlation of leptin with remnant cholesterol in all the groups in the regression analysis. Hence, there was no significant positive association between leptin and remnant cholesterol; this can be explained in the context of a higher level of remnant cholesterol, which is not solely driven by adiposity and body mass index.

Ethical Statement

Ethical approval was obtained from the Ethics Committee (NAUTH/CS/66/VOL.11/ 154/2018/088) of Nnamdi Azikiwe University Teaching Hospital Nnewi, and informed consent was sought from participants.

Conflict of Interest

The author declares no conflict of interest.

Acknowledgements

We thank all the participants of the present study for their patience and commitment throughout this research.

Reference

- Barnes LA, Opitz JM, Gilbert-Barnes E. "Obesity: genetic, molecular, and environmental aspects". *American Journal of Medical Genetics. Part A*. 143A 2007(24):3016–34
- World Health Organization Expert Committee: Physical Status; the Use and Interpretation of Anthropometry. Report of a WHO Expert Committee. Technical Report Series 854. Geneva: WHO 1995
- Bakari AG, Onyemelukwe GC, Sani BG, Aliyu IS, Hassan SS, Aliyu TM. Obesity, overweight and underweight in suburban northern Nigeria. *International Journal of Diabetes Metabolism*. 2007;15:68-9
- Patel M, Patel IM, Patel YM, Rathi SK. A hospital-based observational study of type 2 diabetic subjects from Gujarat, India. *Journal of Health Population and Nutrition* 2011; 29:265-72
- Akintunde AA, Akinwusi PO, Adebayo RA, Ogunyemi S, Opadijo OG. Burden of obesity in essential hypertension: Pattern and prevalence. *Nigerian Journal of Clinical Practice*. 2010;13:399-402
- Amole IO, OlaOlorun AD, Odeigah LO, Adesina SA. The prevalence of abdominal obesity and hypertension amongst adults in Ogbomoso, Nigeria. *African Journal of Primary Health Care. Family Medicine*. 2011;3(1):25-32
- Akarolo-Anthony SN, Willett WC, Spiegelman D, Adebamowo CA. Obesity epidemic has emerged among Nigerians. *BioMedical Central Public Health*. 2014; 14:455
- Iloh GUP, Amadi AN, Nwankwo BO, Ugwu VC. Obesity in adult Nigerians: a study of its pattern and common primary co-morbidities in a rural Mission General Hospital in Imo state, south-eastern Nigeria. *Nigerian Journal of Clinical Practice*. 2011; 14(2):50-59
- Lopez JP, Juan B, Sandra M, Rueda Q, Cristina LL, Camilo F. Obesity and preeclampsia; common pathophysiological mechanism. *Frontiers in physiology*. 2018;9:1838
- Nguyen NT, Magno CP, Lane KT, Hinojosa MW, Lane JS. Association of hypertension, diabetes, dyslipidemia, and metabolic syndrome with obesity: Findings from the national health and nutrition examination survey, 1999 to 2004. *Journal of American College of surgeons*. 2008;207:928-34
- Considine R.V, Sinha M.K, Heiman M.L, Kriauciunas A, Stephens T.W, Nyce M.R, Ohannesian J.P, Marco C.C, McKee L.J, et al. Serum immunoreactive-leptin concentrations in normal-weight and obese humans. *New England Journal of Medicine*. 1996;334, 292–295
- Boden WE, Probstfield JL, Anderson T, Chaitman BR, Desvignes-Nickens P, et al. Niacin in patients with low HDL cholesterol levels receiving intensive statin therapy. *New England Journal of Medicine*. 2011;365:2255-67
- Chu NF, Stempfer MJ, Spiegelman D, Rifai N, Hotamisliligil ES, Rimm EB. Dietary and lifestyles factors in relation to plasma leptin concentration among normal weight and overweight men. *International journal of obesity and related metabolic disorders*. 2001;25(1):106-14
- Janeckova R. The role of leptin in human physiology and pathophysiology. *Physiological Research/ Academia Scientarium Bohemoslovaca*. 2002;50(5):433-59
- Lahlou N, Tarik I, Lebouc Y, Jean-Claude C, Camoin L, Marc R, Girard J. Mutations in the human leptin and leptin receptor genes as model of serum leptin receptor regulation. *Diabetes*. 2002;51(6):1980-1982
- Dallinger-Thie GM, Kroon J, Chapman MJ. Triglyceride-rich lipoproteins and Remnants: Targets for Therapy? *Current Cardiology Reports*. 2016;18(7):67-74
- Goliash G, Wiesbauer F, Blessberger H, Demyanets S, Wojta J, Huben K, Maurer G, Schillinger M, Speidl WS. "Premature myocardial infarction is strongly associated with increased levels of remnant cholesterol". *Journal of Clinical Lipidology*. 2015;9(6):801-806
- Masuoka H, Kamei S, wagayama H, Ozaki M, Kawasaki A, Tanaka T, Kitamura M, Katoh S, Shintani U, Misaki M, Sugawa M, Ito M, Nakano T. "Association of remnant-like particle cholesterol with coronary artery disease in patients with normal cholesterol total cholesterol levels". *American Heart Journal* 2000;139(2):305-310
- Varbo A, Benn M, Davey Smith G, Timpson NJ, Tybjaerg-Hansen A, Nordestgaard BG. Remnant cholesterol, low density lipoprotein cholesterol, and blood pressure as mediators from obesity to ischemic heart disease. *Circulation Research*. 2015;116:665–73
- Allen CC, Poon LS, Chan CS, Richmond W, Fu PC. Enzymatic determination of total Serum Cholesterol. *Clinical chemistry*. 1974;20:470-475
- Trinder P. Determination of Glucose in Blood Using Glucose Oxidase with an Alternative Oxygen Acceptor. *Annals of clinical biochemistry*. 1969;21:6-24
- Friedwald WT, Levy RI, Fredrickson DS. Estimation of the concentration of lowdensity lipoprotein cholesterol in plasma without use of the preparative ultracentrifuge. *Clinical Chemistry*. 1972;18(6):499-502
- Sone M, Osamura RY. Leptin and the pituitary. *Pituitary*. 2001;4:15-23

24. Chubenko E, Belyaeva O, Bolshakova O, Berkovich O, Bazhenova E. Leptin concentration in obese and hypertensive patients. *Journal of hypertension*. 2010;28:190-12-465
25. Okpara H, Osege I, Azinge E. Relationship between serum leptin and insulin resistance among obese Nigerian women. *Annals of African Medicine*. 2016;15:14-19
26. Masoud Y Al-Maskari, Adel A Al-naqdy. Correlation between serum leptin levels, body mass index and obesity in Omanis. *Sultan Qaboos University Medical Journal*. 2006;6:27-31
27. Varbo A, Freiberg JJ, Nordestgaard BG. "Remnant cholesterol and myocardial infarction in normal weight, overweight, and obese individuals from Copenhagen General Population Study". *Clinical Chemistry*. 2018;64(1):219-230

Contact Details

Chinonso J Nnamdi

Chemical Pathology, Faculty of Basic Clinical Sciences,
Nnamdi Azikiwe University, Awka, Nigeria
E-mail: cj.nnamdi@unizik.edu.ng
ORCID: 0000-0003-3795-1275

Amah K Ubuo

Chemical Pathology, Faculty of Basic Clinical Sciences,
Nnamdi Azikiwe University, Awka, Nigeria
E-mail: amahubuo@yahoo.com
ORCID: 0000-0001-7911-7436

Anaelechi J Onuegbu

Chemical Pathology, Faculty of Basic Clinical Sciences,
Nnamdi Azikiwe University, Awka, Nigeria
E-mail: ajonuegbu@yahoo.com
ORCID: 0000-0001-7342-9949

Madu J Olisekodiaka

Clinical Chemistry Unit, Department of Biomedical
Sciences, College of Health Sciences, Ladoke Akintola
University of Technology, Ogbomoso
E-mail: edenjaph@yahoo.ca
ORCID: 0000-0003-2487-1233

Chima H Okpara

Department of Chemical Pathology, University of
Calabar, University of Calabar Teaching Hospital,
Calabar, Nigeria,
Email: okphenchi@yahoo.com
ORCID: 0000-0003-4587-0504

Ekenedirichukwu J Okwara

Chemical Pathology, Faculty of Basic Clinical Sciences,
Nnamdi Azikiwe University, Awka, Nigeria
E-mail: igwechidi@yahoo.com
ORCID: 0000-0003-1477-1992

Adamma R Analike

Chemical Pathology, Faculty of Basic Clinical Sciences,
Nnamdi Azikiwe University, Awka, Nigeria
E-mail: rose_analyke@yahoo.com
ORCID: 0000-0002-4786-379X

Chidiadi M Njoku

Chemical Pathology, Faculty of Basic Clinical Sciences,
Nnamdi Azikiwe University, Awka, Nigeria
E-mail: cj.nnamdi@unizik.edu.ng
ORCID: 0000-0003-2837-5368

How to cite?

Nnamdi CJ, Ubuo AK, Onuegbu AJ, Olisekodiaka MJ, Okpara CH, Okwara EJ, Analike AR, Njoku CM. Remnant Cholesterol and Leptin: Any Association in Normal Weight, Over Weight and Obese Individuals in South East Nigeria. *Ulutas Med J*. 2022;8(4):135-141

DOI: 10.5455/umj.20220616022839