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Serum lipid profile based on the prandial state among adult subjects

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This study was aimed to access the variations in serum lipid parameters based on the prandial state of adult patients. Through a cross-sectional descriptive and analytical study conducted in Parakou (Benin), from February 1 to May 1, 2020, 100 adult subjects (average age 31.21±12.36 years) were selected with systematic census. After ethical advice, a first sample was taken on empty-bellied adult subjects. Then, three additional samples were respectively taken two hours, four hours, and six hours after these subjects had eaten a meal of their choice. Serum lipid parameters were measured through enzymatic colorimetric methods. The average values of fasting lipid parameters in g/L were: Total cholesterol (1.48±0.35), HDL cholesterol (0.49±0.14), LDL cholesterol (0.84±0.36) and triglycerides (0.63±0.34). The comparison of the fasting and postprandial lipid parameters values is presented as follow: Triglyceridemia was significantly higher 2 h (P=0.000) and 4 h after meal (P=0.001). Only one type of food consumed was associated with mixed hyperlipidemia at six hours postprandial state (P=0.022). It may therefore be concluded that consumed foods do not cause most dyslipidemias 6 h in the postprandial state. During the screening for dyslipidemias in adults, the lipid profile can therefore be measured after six hours postprandial state.

Key words: Lipid profile, fasting, prandial state, adults.

INTRODUCTION

Serum lipid profile is commonly determined for the cardiovascular risk prediction. It is a routine medical examination that helps perform the screening of dyslipidemias (Nigam, 2011). Dyslipidemia is an important risk factor for coronary artery disease and stroke.

The joint consensus statement from the European Atherosclerosis Society and European Federation of Clinical Chemistry and Laboratory Medicine recommend several conditions for lipid test: i) non-fasting in most patients, including initial lipid profile testing in any patient, for cardiovascular risk assessment, patients admitted with acute coronary syndrome, in children, if preferred by the patient, in diabetic patients (due to hypoglycaemic risk), in the elderly and patients on stable drug therapy; ii) fasting can sometimes be required if non-fasting triglycerides >5 mmol/L (440 mg/dL), known hypertriglyceridaemia followed in lipid clinic, recovering from hypertriglyceridaemic pancreatitis, starting

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medications that cause severe hypertriglyceridaemia, additional laboratory tests are requested that require fasting or morning samples (Catapano et al., 2016).

For long, fasting blood samples have been considered as a standard for the measurement of lipid parameters after 8 to 12 h fasting (Stone et al., 2014; Driver et al., 2016). In their physiology, human beings do not adhere to that fasting time limit on a daily basis. Many studies have reported that the benefits of fasting lipid tests are not higher than those done in a non-fasting state (Nordestgaard, 2017; Pati and Singh, 2017; Scartezini et al., 2017). Well-conducted prospective studies found similar associations with cardiovascular risk by using lipid profile in the fasted state as well as in the non-fasting state (Mora et al., 2008; Langsted et al., 2008; Di Angelantonio et al., 2009; Doran et al., 2014).

To date, there is no scientific evidence explaining why fasting should be better than non-fasting during the lipid test assessment for the prediction of cardiovascular risk. Several clear benefits of non-fasting rather than fasting samples for lipid parameters' determination, may be mentioned: (i) blood collection in laboratory is simplified; (ii) the inconveniences related to fasting are avoided for the benefit of the patients; (iii) the risk for hypoglycemia due to fasting is minimized for subjects with diabetes (Anderson et al., 2016; Catapano et al., 2016; Jellinger et al., 2017; Scartezini et al., 2017).

Lipid parameters' determination according to the prandial state has been reported in literature. No difference was observed between fasting and non-fasting values of total cholesterol (TC), HDL cholesterol (HDL-C), and LDL cholesterol (LDL-C) whereas triglyceridemia (TG) was significantly higher in the postprandial state among diabetic patients (Gupta et al., 2016). A substantial increase in postprandial triglyceridemia among subjects with dyslipidemia on drug therapy and also among untreated subjects was found (Abdel-Aziza et al., 2017). The ASCOT-LLA study carried out on 8270 patients in the postprandial state, revealed a moderate increase in triglyceridemia and marginal change in HDL-C, LDL-C and TC compared to fasting subjects (Mora et al., 2019). In an observational study, non-fasting triglyceridemia is approximately 20% higher on average than the one measured in the fasted state; although the magnitude of the difference is subject to substantial interpatient variability (Rahman et al., 2018).

Many countries are currently revising their guidelines, seeking a consensus on lipid profile measurement for the prediction of cardiovascular risk in the non-fasting state (Anderson et al., 2016; Catapano et al., 2016; Jellinger et al., 2017; Scartezini et al., 2017). In Africa, and more particularly in Benin, lipid profile measurement in the fasted state is always adopted despite frequency of examination request for the exploration of cardiovascular diseases (CVDs). In Benin, to the best of our knowledge, no study has been conducted in order to reduce patients' constraints for the performance of lipid profile test, to get results in the physiological condition, and to assist the clinician in making a quick decision. This research work aimed to investigate variations in serum lipid parameters based on the prandial state among adult subjects in order to consider doing non-fasting lipid profile test.

MATERIALS AND METHODS

Type and period of study

This research work was a descriptive cross-sectional study with analytical purpose; the data used were collected over a three-month period running from February 1 to May 1, 2020.

Study target population

It consisted of adult subjects from both sexes living in the city of Parakou (Republic of Benin). This study included subjects, healthy or not, aged 18 years and more, who gave their writing informed consent to participate to the study. Pregnant and breastfeeding women were excluded.

Sampling

The authors did a systematic census of all adult subjects who volunteered to participate in this study during the data collection period, and meeting our inclusion criteria, in Parakou Teaching Hospital. This study involved 100 adult subjects.

Study variables

The dependent variable was lipid profile according to the fasting and prandial state. The independent variables were sociodemographic, anthropometric parameters and type of food consumed.

Data collection

After the administration of questionnaire and measurement of anthropometric parameters, a first venous blood sample was collected into a dry tube in fasting for at least 12 h. On the next day, subjects were admitted to Parakou Teaching Hospital. Following an overnight fast of at least 12 h, subjects were provided with a food of their choice. Venous blood was sampled at 2, 4 and 6 h following the food intake. The types of foods consumed include:

-Type 1 food: rice + side dish (fry, tomato sauce, cheese, egg);
-Type 2 food: flour paste (maize, dried cassava chips) + sauce (tomato or vegetable) or pounded yam + sauce as side dish (tomato, groundnut, vegetable or gluey sauce) or *akassa* (that is a cooked, ground and soaked maize paste) + side dish (onion, tomato pepper juice);
-Type 3 food: porridge (maize, millet) + wheat doughnuts or fritters;
-Type 4 food: alimentary paste (spaghetti, couscous) or bread + sandwich; biscuits; cake;
-Type 5 food: beans + side dish (groundnut oil or palm oil, cassava flour).

The blood samples (4 mL) collected were centrifuged at 1500 g for

Table 1. General characteristics of study target population.

Parameter	Mean values±SD			
	Total	Male (n=58)	Female (n=42)	P#
Age (years)	31.21±12.36	32.31±12.34	29.69±12.39	*0.298*
BMI (kg/m^2)	23.97±4.56	23.18±3.95	25.07±5.13	0.040
SBP (mmHg)	116.61±21.86	119.33±25.44	112.86±15.14	0.145
DBP (mmHg)	75.06±14.67	74.22±16.24	76.21±12.29	0.506

N=100. BMI: body mass index; SBP: systolic blood pressure; DBP: diastolic blood pressure; SD: standard deviation; #: Student's-t independent.

Table 2. Distribution of mean values of fasting serum lipid parameters (in g/L) according to subjects' sex.

Parameter	Total	Male (n=58)	Female (n=42)	P#
Total cholesterol	1.49±0.35	1.44±0.35	1.55±0.36	0.137
HDL cholesterol	0.50±0.15	0.48±0.16	0.53±0.12	0.082
LDL cholesterol	0.85±0.37	0.82±0.38	0.89±0.35	0.322
Triglycerides	0.70±0.35	0.74±0.39	0.64±0.27	0.176

N=100. #Student's-t independent.

5 min, and then serums were decanted. The latter were used on the same day to measure the lipid parameters.

Total cholesterol was measured through endpoint enzyme assay with cholesterol oxidase (MacLachlan et al., 2000), HDL cholesterol measured using phosphotungstic acid-magnesium precipitation procedure (Warnick et al., 1979), and triglycerides measured using endpoint enzyme assay with glycerophosphate oxidase (Solera, 2000). LDL cholesterol was calculated with the formula of Friedewald et al. (1972) under the conditions of triglyceridemia lower than 3.5 g/L.

Data analysis

Statistical analyses were performed with the SPSS software (2011, IBM Corporation). Analysis of variance (ANOVA) and Student's t independent test were used to determine if differences in fasting and non-fasting measurements for total cholesterol, LDL cholesterol, HDL cholesterol and triglycerides were statistically significant. Chi-square independence test of Pearson was used to determine the association between type of food and dyslipidemia. Significance level was set at 0.05.

Ethical considerations

The research protocol of this study was approved by the Local Ethics Committee for Biomedical Research of the University of Parakou (Opinion No. 0302/CLERB-UP/P/SP/R/SA).

RESULTS

General characteristics of study target population

The number of participant was 100, and 58% of them were men (sex-ratio = 1.38). Their mean age was 31.21±12.36 years and mean body mass index (BMI) was 23.97±4.56kg/m2. Mean systolic blood pressure was 116.61±21.86 mmHg and their mean diastolic blood pressure was 75.06±14.67 mmHg (Table 1). Women had a significantly higher body mass index (P=0.040).

Fasting serum lipid parameters

Mean values of fating serum lipid parameters were presented in Table 2. There was no significant difference of fasting serum lipid parameters according to the sex (P>0.05).

Postprandial lipid parameters' values

Table 3 shows the comparison of fasting and postprandial serum lipid parameters among male subjects. The levels of triglycerides after meal were increased compared to fasting triglycerides values, and their changes were statistically significant at 2 h (P=0.000) and 4 h (P=0.004), when analyzed using student's t- independent test. The highest levels of triglycerides were noted 2 h after meal (1.12±0.58 g/L). There was no significant difference between fasting and non-fasting serum lipid parameters among female subjects (P>0.05) when analyzed using both student's t- independent and ANOVA test (Table 4).

Table 5 shows the comparison of fasting and postprandial serum lipid parameters among all the 100 subjects. Except for triglycerides, there were no substantial changes in the distributions of lipid parameters as a function of time since meal intake.

Table 3. Comparison of mean values of fasting and postprandial serum lipid parameters (in g/L) among male subjects.

Parameter	T0 MV±SD	T2 MV±SD	T4 MV±SD	T6 MV±SD	P*	$P(T_0;T_2)^{\#}$	$P(T_0;T_4)^{\#}$	$P(T_0;T_6)^{\#}$
TC	1.44±0.35	1.52±0.46	1.51±0.49	1.51±0.47	*0.774*	*0.322*	*0.382*	*0.375*
HDLC	0.48±0.16	0.44±0.16	0.44±0.14	0.45±0.15	*0.532*	*0.230*	*0.205*	*0.311*
TG	0.74±0.39	1.12±0.58	0.99±0.51	0.86±0.48	*0.000*	*0.000*	*0.004*	*0.122*
LDLC	0.82±0.38	0.84±0.37	0.88±0.42	0.89±0.40	*0.736*	*0.768*	*0.432*	*0.315*

N=58. TC: Total cholesterolemia; HDLC: HDL cholesterolemia; LDLC: LDL cholesterolemia; TG: Triglyceridemia; MV±SD: Mean value±Standard deviation; $P(T_0;T_2)$: P-value of comparison of the mean values of fasting serum lipid parameters and two hours after meal; $P(T_0;T4)$: P-value of comparison of the mean values of fasting serum lipid parameters and four hours after meal; $P(T_0;T6)$: P-value of comparison of the mean values of fasting serum lipid parameters and six hours after meal.
*: ANOVA test; $^{\#}$: Student's-t independent.

Table 4. Comparison of fasting and postprandial serum lipid parameters (in g/L) among female subjects.

Parameter	T0 MV±SD	T2 MV±SD	T4 MV±SD	T6 MV±SD	P*	$P(T_0;T_2)^{\#}$	$P(T_0;T_4)^{\#}$	$P(T_0;T_6)^{\#}$
TC	1.55±0.36	1.54±0.32	1.51±0.37	1.52±0.38	*0.957*	*0.878*	*0.617*	*0.709*
HDLC	0.53±0.12	0.51±0.10	0.51±0.11	0.50±0.11	*0.608*	*0.543*	*0.400*	*0.205*
TG	0.64±0.27	0.79±0.43	0.75±0.42	0.67±0.35	*0.229*	*0.067*	*0.144*	*0.715*
LDLC	0.89±0.35	0.87±0.29	0.85±0.34	0.89±0.36	*0.932*	*0.717*	*0.582*	*0.971*

N=42. TC: Total cholesterolemia; HDLC: HDL cholesterolemia; LDLC: LDL cholesterolemia; TG: Triglyceridemia; MV±SD: Mean value±Standard deviation; P(T0;T2): P-value of comparison of the mean values of fasting serum lipid parameters and two hours after meal; $P(T_0;T4)$: P-value of comparison of the mean values of fasting serum lipid parameters and four hours after meal; $P(T_0;T6)$: P-value of comparison of the mean values of fasting serum lipid parameters and six hours after meal. *: ANOVA test; $^{\#}$: Student's-t independent.

Table 5. Comparison of mean values of fasting and postprandial serum lipid parameters (in g/L) among all study subjects.

Parameter	T0 MV±SD	T2 MV±SD	T4 MV±SD	T6 MV±SD	P*	$P(T_0;T_2)^{\#}$	$P(T_0;T_4)^{\#}$	$P(T_0;T_6)^{\#}$
TC	1.49±0.35	1.53±0.41	1.51±0.44	1.51±0,43	*0.925*	*0.472*	*0.681*	*0.629*
HDLC	0.50±0.15	0.47±0.14	0.47±0.13	0.47±0,13	*0.352*	*0.187*	*0.135*	*0.129*
TG	0.70±0.35	0.98±0.54	0.89±0.49	0.78±0,44	*0.000*	*0.000*	*0.001*	*0.135*
LDLC	0.85±0.37	0.85±0.34	0.87±0.38	0.89±0,38	*0,847*	*0.981*	*0.762*	*0.439*

N=100. TC: Total cholesterolemia; HDLC: HDL cholesterolemia; LDLC: LDL cholesterolemia; TG: Triglyceridemia; MV±SD: Mean value±Standard deviation; $P(T_0;T_2)$: P-value of comparison of the mean values of fasting serum lipid parameters and two hours after meal; $P(T_0;T4)$: P-value of comparison of the mean values of fasting serum lipid parameters and four hours after meal; $P(T_0;T6)$: P-value of comparison of the mean values of fasting serum lipid parameters and six hours after meal.
*: ANOVA test; $^{\#}$: Student's-t independent.

Triglycerides increased after meal among all subjects of the study; the highest levels of triglycerides were noted 2 h after meal (0.98±0.54). Triglyceridemia was significantly higher 2 h (0.70±0.35 g/L vs 0.98±0.54 g/L; P=0.000; 40% increased) and 4 h (0.70±0.35 g/L vs 0.89±0.49 g/L; P=0.001; 27% increased) in postprandial state when analyzed using student's t- independent test. ANOVA test showed significant difference between mean values of triglyceridemia in fasting (0.70±0.35 g/L), 2 h (0.98±0.54 g/L), 4 h (0.89±0.49 g/L) and 6 h (0.78±0,44 g/L) after meal (P=0.000).

The association between types of food and dyslipidemias 6 h after meal was presented in Table 6.

Six hours after meal, type 1 food (rice + side dish (fry, tomato sauce, cheese, egg)) was associated with mixed hyperlipidemia (P = 0,022) when analyzed using chi-

Table 6. Distribution of dyslipidemias according to foods consumed 6 h after meal.

Type of food	Dyslipidemias six hours after meal						
	Total N=100	THC N=14	HDLH N=32	LDLH N=14	HTG N=9	MHL N=5	AD N=4
	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)
Type 1 food							
Yes	20(20.00)	3(21.43)	6(18.75)	2(14.29)	3(33.33)	3(60.00)	0(0.00)
No	80(80.00)	11(78.57)	26(81.25)	12(85.71)	6(66.67)	2(40.00)	4(100.00)
$P^{§}$	-	0.885	0.830	0.564	0.295	0.022	0.307
Type 2 food							
Yes	18(18.00)	2(14.29)	8(25.00)	2(14.29)	2(22.22)	1(20.00)	2(50.00)
No	82(82.00)	12(85.71)	24(75.00)	12(85.71)	7(77.78)	4(80.00)	2(50.00)
$P^{§}$	-	*0.696*	*0.211*	*0.696*	*0.730*	*0.905*	*0.089*
Type 3 food							
Yes	23(23.00)	3(21.43)	7(21.88)	5(35.71)	1(11.11)	0(0.00)	1(25.00)
No	77(77.00)	11(78.57)	25(78.13)	9(64.29)	8(88.89)	5(100.00)	3(75.00)
$P^{§}$	-	*0.880*	*0.854*	*0.223*	*0.374*	*0.210*	*0.923*
Type 4 food							
Yes	23(23.00)	5(35.71)	8(25.00)	5(35.71)	1(11.11)	1(20.00)	0(0.00)
No	77(77.00)	9(64.29)	24(75.00)	9(64.29)	8(88.89)	4(80.00)	4(100.00)
$P^{§}$	-	*0.223*	*0.744*	*0.223*	*0.374*	*0.870*	0.265
Type 5 food							
Yes	6(6.00)	1(7.14)	2(6.25)	1(7.14)	0(0.00)	0(0.00)	0(0.00)
No	94(94.00)	13(92.86)	30(93.75)	13(92.86)	9(100.00)	5(100.00)	4(100.00)
$P^{§}$	-	*0.846*	*0.942*	*0.846*	*0.427*	*0.562*	*0.606*

THC: Total hypercholesterolemia; HDLH: HDL hypocholesterolemia; LDLH: LDL hypercholesterolemia; HTG: Hypertriglyceridemia; MHL: Mixed hyperlipidemia; AD: Atherogenic dyslipidemia; §: Chi-square test of Pearson.

square test of Pearson.

DISCUSSION

The results of this study showed that there was no significant difference between the values of total cholesterol, HDL cholesterol and LDL cholesterol (P>0.05) when compared fasting and postprandial lipid parameters. However, there would be a significant difference between fasting and non-fasting triglyceridemia at 2 h (P=0.000) and 4 h (P=0.001).

Other recent research works showed the same results. In the study of Feres et al. (2018), meal did not influence total cholesterol, HDL cholesterol and LDL cholesterol, but triglycerides increased significantly after meal : 2 h (156.0±86.4 mg/dL, P=0.000), 3 h (148.5±92.0 mg/dL, P=0.000) and 4 h (143.4±93.0 mg/dL, P= 0.000). Among hemodialysis patients, post-prandial triglycerides was significantly raised both in male subjects (1.47±0.99 versus 1.67±1.22 mmol/L, P=0.015) and females (1.56±0.08 versus 1.83±0.11 mmol/L, P=0.001) (Alsaran et al., 2009). An Indian study conducted from 2012 to 2014 concluded that triglycerides were significantly increased till 6 to 7 h after meal (Gupta et al., 2016).

Another study mentioned a significant difference in plasma triglyceridemia which has already increased one hour after the last meal and remained high until 7 h later (Nordestgaard et al., 2009). A considerable difference between fasting and non-fasting triglycerides was noted by Pati and Singh (2017). Rahman et al. (2018) observed a non-fasting triglycerides increased by approximately 20%.

In contrast to above-mentioned studies**,** in a survey conducted among subjects with type 2 diabetes who consumed a standardized meal, Lund et al. (2011) highlighted that total cholesterol and HDL cholesterol mean concentrations have each decreased by approximately 0.1 mmol/L in the postprandial state, whereas triglycerides' mean concentrations rose by 0.8

mmol/L; on the contrary, LDL cholesterol considerably declined (P=0.005).

Postprandial increase in triglycerides may be due to their metabolism. In the postprandial state, when metabolic demands are met, the excess energy resulting from foods is directed towards the synthesis of triglycerides and lipogenesis. In the fasted state, the lipids stored as triglycerides might be used to provide energy through the β-oxidation of fatty acids**,** and to ensure gluconeogenesis (Harvey and Ferrier, 2011). According to Kolovou et al. (2019), overproduction and decreased catabolism of triglycerides-rich remnant lipoproteins are the two main mechanisms leading to postprandial lipid and lipoprotein abnormalities. Triglycerides-rich remnant lipoproteins are a spectrum of particles, some of which are almost as small as LDL particles although most remnant particles are larger and have a lower potential to traverse endothelial cells than the smaller LDL particle. On the other hand, triglycerides-rich remnant lipoproteins are 5-20 times richer in cholesterol content compared with an LDL particle and because of the larger size of triglycerides-rich remnant lipoproteins compared with the smaller size of LDL particles, once they have penetrated into the intima, they may remain there for longer. Other factors influencing lipid profile results may explain increased triglycerides in the postprandial state: meals containing fats, alcohol consumption during or before meal, and other macronutrients (Bae et al., 2003; Mora et al., 2008).

Six hours after meal, only type 1 food was associated with mixed hyperlipidemia (P=0,022). The composition of the type 1 food containing the accompanying sauce, which is richer in fats than the 4 other foods, and eggs could raise cholesterol and triglycerides found in mixed hyperlipidemia; however this observation remains to be confirmed. Lipid concentrations vary widely after meals depending on the postprandial time and the type of food intake. Individual responses to food ingestion are extremely heterogeneous. In addition, there are cultural differences to food ingestion. Standardization is key to establishing a robust laboratory test. For each non-fasting condition, a clinical laboratory should have reference values established for several factors other than the lipid profile (Lund and Jensen, 2011).

Standardizing the procedures and the patient preparation for a fasting protocol is hard enough, and the problem becomes even more complex with a non-fasting protocol (Lorenzo Lozano et al., 2017). A recent American Heart Association statement on hypertriglyceridemia and coronary heart disease suggests that clinicians can use a non-fasting triglycerides level of >200 mg/dL to identify hypertriglyceridemic states (Miller et al., 2011). Among normotriglyceridemic subjects (that is, fasting triglycerides levels <150 mg/dL), consumption of a low-fat breakfast (typically <15 g of fat) before blood sampling should not induce an increase in postprandial triglycerides levels by more than 20%, and is unlikely to cause levels to exceed 200 mg/dL (Dubois et al., 1998). Additional more recent data have suggested that a nonfasting triglycerides level of 175 mg/dL could also be reasonable (White et al., 2015). Follow-up fasting triglycerides testing in these cases are not needed, but this should not dissuade further discussion of lifestyle measures.

The authors hypothesized that there was no difference in fasting and non-fating serum lipid parameters in adult subjects. Our findings show that levels of lipids parameters after normal food intake differ only minimally from levels in the fasting state. It may therefore be concluded that consumed foods do not cause most dyslipidemias during 6 hours in the postprandial state. As in studies conducted by Nordestgaard (2017), Mora et al. (2019) and Yang et al. (2020), the fasting state is not necessary for the screening of dyslipidemias among Benin adult subjects, given the wide variety of foods consumed. From above findings we can affirm that non-fasting blood draws may be highly effective and practical for lipid profile testing.

Conclusion

This research work, focused on variations in serum lipid parameters depending on the prandial state among adult subjects, points out that total cholesterolemia, HDL cholesterolemia and LDL cholesterolemia do not significantly vary whatever the prandial state is. However triglyceridemia significantly raised until four hour in the postprandial state with return to normal six hour later. Only food rich in fats consumed is associated with mixed hyperlipidemia, six hours after meal. It is recommended that non-fasting blood samples be routinely used for the assessment of plasma lipid profiles after a 6-h fasting among adult subjects.

CONFLICT OF INTERESTS

The authors have not declared any conflict of interests.

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