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Influence of a Regular, Standardized Meal on Lipid Profile of People with Diabetes

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Abstract

Aims: The need to fast before lipid screening has been questioned in the past years. The aim of this study was to determine the influence of a light meal on the lipid profile in people with diabetes.

Methods: 115 participants with type 2 diabetes were recruited between April/2013-August/2014 from our Outpatient Diabetes Education Clinic. Clinical and analytical evaluation took place in 2 moments (8-hour fasting=t_0; 2h after a light standardized meal=t_1), with measurements of total cholesterol, LDL-cholesterol, HDL-cholesterol and triglycerides.

Results: Triglycerides concentration increased between the 2 moments (median difference t_1-t_0=0.07 mmol/L, p=0.002) but the total cholesterol, LDL-cholesterol, HDL-cholesterol and non HDL- cholesterol did not change significantly. Performing an analysis according to the LDL-cholesterol therapeutic goals proposed by Adult Treatment Panel III, we found an agreement between fasting and postprandial assessments of 91.1% for the goal of 2.6 mmol/L (102/112), and of 97.3% for the goal of 1.8 mmol/L (109/112). The same analysis was performed for the secondary goal, non HDL-cholesterol.

Conclusion: The data presented suggest that the nonfasting lipid profile can be an alternative to the fasting lipid profile in selected patients. Larger studies are needed to confirm these results and demonstrate an association of nonfasting lipemia and cardiovascular risk in individuals with diabetes.

Keywords: Diabetes; Lipid metabolism; Fasting

Abbreviations:

ATP III: Adult Treatment Panel III; HDL-C: HDL Cholesterol; LDL-C: LDL Cholesterol; nonHDL-C: non-HDL Cholesterol; TG: Triglycerides; TC: Total Cholesterol

Introduction

The plasma concentration of LDL cholesterol (LDL-C) is a recognized cardiovascular disease risk factor [1-3]. The studies that address this question are mainly based on fasting samples, and these are the current recommendations of most international societies [1,2,4-7].

Since humans spend most of their time in the nonfasting state and the postprandial lipemia has shown to be useful in the cardiovascular disease risk prediction [8-10], the need to fast before lipid screening has recently been questioned [8,11,12]. Moreover, nonfasting levels of lipids seem to differ only minimally from levels in the fasting state, both in the general population [8,13] and in individuals with diabetes [11]. Allying these facts to the practical benefit of measuring the lipid profile in the postprandial state, the Danish national recommendation is, since 2009, for nonfasting assessment of LDL-C [14].

The aim of our work was to determine the influence of a light meal on the lipid profile in individuals with diabetes.

Methods

A total of 115 consecutive participants with type 2 diabetes were included, recruited between April/2013 and August/2014 from the Outpatient Diabetes Education Clinic of Hospital de Santo António, Centro Hospitalar do Porto. All participants were similarly evaluated. Blood samples were drawn in 2 moments for measurements of total cholesterol (TC), LDL cholesterol, HDL cholesterol (HDL-C) and triglycerides (TG). The first blood sample was collected between 08:30 and 09:00 am after an 8 hour overnight fast (t_0) and the second blood sample was collected 2 hours after a light meal (t_1), containing standardized amounts of carbohydrates, protein and lipids. This meal included bread with ham or butter and coffee with milk (240-250 kcal; fat -l 7 g, protein - 9-12 g and carbohydrates -l 34 g). No food or drinks, apart from water, were allowed to be ingested between the two blood collections. The measurements were made in the same laboratory, by standard hospital assays - CT, HDL-C e TG through enzymatic methods (Roche) in freshly drawn lithium-heparin plasma samples. LDL-C was calculated using the Friedewald Equation (LDLC=TC-HDLC-[TG/5]) if TG levels <400 mg/dl and measured directly at higher levels [15]. Non-HDL cholesterol (nonHDL-C) was also calculated (TC-HDLC). Because of its influence on lipid profile, we excluded thyroid dysfunction in all participants through TSH

measurement by electrochemiluminescence at t_0. Duration of diabetes was self-reported; hypertension was self-reported disease or use of antihypertensive medication; smokers were active smokers. Written informed consent was obtained from all patients.

The analysis of the data was conducted by using SPSS v20. LDL-C and nonHDL-C levels showed a normal distribution, unlike TC, TG and HDL-C. Comparison between the 2 moments were carried out by use of paired samples t-test (LDL-C and nonHDL-C) and Wilcoxon test (TC, TG, HDL-C). Paired LDL-C samples of participants with TG levels above 400 mg/dl were excluded from the analysis (n=3).

Furthermore, we analyzed the data according to the Adult Treatment Panel III (ATP III) fasting LDL-C goals: we investigated the proportion of participants with LDL-C at $t_0 \geq 2.6$ mmol/L and LDL-C at $t_1<2.6$ mmol/L and vice versa. We made a similar analysis using the cutpoint of 1.8 mmol/L and the secondary goal, nonHDL-C (1).

Results

The characteristics of the individuals included in the study are shown in Table 1. TG concentration increased between the 2 moments (median difference t_1-t_0=0.07 mmol/L, p 0.002) but the TC, HDL-C, LDL-C and non-HDL-C did not change significantly (Table 2).

Female (%)	**50.4%**
Age (years)	57 (13; 40-79)
Duration of diabetes (years)	8.0 (11; 1-38)
HbA1c (%,mmol/mol)	8.3% (2.7; 4.8-14.0); 67 mmol/mol (9; 29-130)
Hypertension (%)	75%
Smoking habits (%)	8.8%
Body mass index (kg/m2)	29.0 (5.2; 19.3-44.0)
Statin therapy (%)	66.3%
Fibrate therapy (%)	17.3%

Table1: Characteristics of the study population. Continuous variables are shown as median (interquartile range, minimum-maximum).

	Fasting measurement (t_0)	**Postprandial measurement (t_1)**	**Difference t_1-t_0**	**p**
TC (mmol/L)ǂ	4.4 (1.2; 2.4-7.3)	4.4 (1.2; 2.4 - 7.3)	0.0 (0.2; -0.6 - 0.5)	0.74
TG (mmol/L)ǂ	1.4 (0.8; 0.4-4.5)	1.5 (0.9; 0.5-4.2)	0.1 (0.3; -1.1 - 0.9)	0.002
HDL-C (mmol/L)ǂ	1.2 (0.5; 0.4-2.6)	1.2 (0.5; 0.4-2.4)	0.0 (0.1; -0.3 - 0.2)	0.77
LDL-C (mmol/L)*	2.5 (0.8)	2.5 (0.8)	-0.03 (0.2)	0.06
nonHDL-C (mmol/L)*	3.3 (0.9)	3.3 (0.9)	0.0 (0.1)	0.89

Table 2: Fasting and 2 hour postprandial lipid values. ǂdata presented as median (interquartile range, minimum-maximum); *data presented as mean (standard deviation).

When we performed the same analysis by gender, the results were similar, except for HDL-C in men, where HDL-C decreased 0.02±0.07 mmol/L (p 0.03). We also analyzed the results according to the use of statins. The results obtained in the subgroup taking statins were similar to those of the overall sample, with an increase of TG concentration between the two moments (median 0.03 mmol/L, interquartile range 0.4 mmol/L, minimum -3.0 mmol/L, maximum 0.9 mmol/L; p 0.03); in the remaining parameters analyzed there were no statistically significant changes (data not shown). Given the small number of participants taking fibrates that sub analysis was not done.

		Postprandial (mmol/L)					**Postprandial (mmol/L)**		
		≥2.6	**<2.6**	**Total**			**≥1.8**	**<1.8**	**Total**
Fasting (mmol/L)	**≥2.6**	49	5	54	**Fasting (mmol/L)**	**≥1.8**	88	3	91
	<2.6	5	53	58		**<1.8**	3	18	21
	Total	54	58			**Total**	91	21	

Table 3: LDL-C according to the 2 therapeutic goals proposed by ATP III.

Table 3 shows the analysis according to the two therapeutic goals for LDL-C proposed by ATP III. We also present the data concerning the secondary goal, nonHDL-C (Table 4).

		Postprandial (mmol/L)					**Postprandial (mmol/L)**		
		≥ 3.4	<3.4	**Total**			≥ 2.6	<2.6	**Total**
Fasting (mmol/L)	≥ 3.4	47	3	50	**Fasting (mmol/L)**	≥ 2.6	85	0	85
	<3.4	3	62	65		<2.6	3	27	30
	Total	50	65			**Total**	88	27	

Table 4: nonHDL-C according to the 2 therapeutic goals proposed by ATP III.

Considering the LDL-C cut-off of 2.6 mom/L, fasting and postprandial assessments agreed on classification for 91.1% of the participants (102/112). The proportion of participants with LDL-C ≥ 2.6 mmol/L was similar in the two assessments (49/54). Postprandial results inappropriately classified participants with borderline values, with small differences between the two measurements (between 0.1 and 0.4 mmol/L). Considering the LDL-C cut-off of 1.8 mmol/L, fasting and postprandial assessments agreed on classification for 97.3% of the participants (109/112). Also here the differences between the two measurements were small (between 0.2 and 0.3 mmol/L).

Regarding the nonHDL-C, and considering the cut-off of 3.4 mmol/L, fasting and postprandial assessments agreed on classification for 94.8% of the participants (109/115). The proportion of participants with discordant results was similar in the two assessments (88/91). Postprandial results inappropriately classified participants with borderline values, with small differences between the two

measurements (between 0.1 and 0.2 mmol/L). Considering the nonHDL-C cut-off of 2.6 mmol/L, fasting and postprandial assessments agreed on classification for 97.4% (112/115). Also in this scenario the differences between the two measurements were small, between 0.1 and 0.3 mmol/L.

Discussion

We found that levels of lipids don't change (TC, HDL-C, LDL-C and nonHDL-C) or change only modestly (TG) in response to a light meal in individuals with diabetes. Statin use or gender did not appear to be influential.

The assumption that the lipid profile varies significantly between the fasting and the nonfasting state is based on studies using oral fat tolerance tests [15,16] and extremely high fat meals [17]. However, both our findings and others' demonstrate that, after a normal meal, the lipid concentrations differ only slightly compared to fasting [8,13,18], even in individuals with diabetes [11], because most people consume much less fat at ordinary meals. It should be considered whether these differences are of clinical importance, especially considering that there are other factors, both analytical and physiological, that also influence levels of lipids [19].

Other argument often presented relates with the calculation of LDL-C using the Friedewald formula, which requires fasting TG measurement. Nonetheless, our results show no significant changes of calculated LDL-C in response to a light meal, despite the increase in TG, as previously demonstrated by Langsted et al [8,11].

In this study, the determination of the lipid profile, including LDL-C and nonHDL-C, in the postprandial period (2 hours after a light meal) allowed a correct classification of most participants in the appropriate risk groups and didn't incorrectly influenced the therapeutic decisions regarding dyslipidemic therapy.

Other of the arguments for the measurement of lipid profile in the fasting period is that the fasting requirement was applied in the majority of lipid-lowering trials [8]. However, more recent studies have focused on cardiovascular disease risk prediction and postprandial lipemia, with no evidence demonstrating that fasting lipid levels are superior to nonfasting levels for cardiovascular risk prediction [8,20]. Indeed, levels of nonfasting TG are as good or even better at predicting future cardiovascular events than levels of fasting TG [9,10,21]. Taking that into account, postprandial lipemia may be masked by measuring lipid profile in the fasting state, and fasting TG concentrations may not be optimal for evaluating cardiovascular risk in these participants [11].

The strengths of our study include using paired samples and a standardized meal, whereas most of previous studies used nonstandardized meals and/or unpaired samples. We do not consider the use of this standardized meal a limitation, since it included ingredients that are part of a regular Western diet. Despite that, we cannot make conclusions regarding other diets. In the same way, we can only draw conclusions about the variation between fasting and 2 hours after eating, because we didn't examine repeated measurements with differing fasting times.

These data point to the possibility of collecting blood samples until 2 hours after a light meal in patients with diabetes. This measure, if implemented, would simplify the blood sampling process, both for patients, practitioners and hospitals. The risk of hypoglycemia in individuals with diabetes with prolonged fasting cannot be neglected, as it is associated with high morbidity [22,23].

Conclusion

The data presented, combined with other recent studies, suggest that the nonfasting lipid profile can be an alternative to the fasting lipid profile in selected patients. Larger studies are needed to confirm these results and to demonstrate an association of nonfasting lipemia and cardiovascular risk in people with diabetes.

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