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Comparison of different methods to investigate postprandial lipaemia

A.J.H.H.M. van Oostrom[1,2], A. Alipour[3], T.P. Sijmonsma[2], C. Verseyden[2], G.M. Dallinga-Thie[4], H.W.M. Plokker[1], M. Castro Cabezas[3*]

[1]Department of Cardiology, St. Antonius Hospital, Nieuwegein, the Netherlands, [2]Department of Vascular Medicine, University Medical Center Utrecht, the Netherlands, [3]Department of Internal Medicine, St. Franciscus Gasthuis, Rotterdam, the Netherlands, [4]Laboratory of Vascular Medicine, Erasmus Medical Center, Rotterdam, the Netherlands, [*]corresponding author: tel.: +31 (0)10-461 72 67, fax: +31 (0)10-461 26 92, e-mail: m.castrocabezas@sfg.nl

ABSTRACT

Postprandial hyperlipidaemia has been associated with coronary artery disease (CAD). We investigated which of the generally used methods to test postprandial lipaemia differentiated best between patients with premature CAD (50±4 years, n=20) and healthy controls. Furthermore, the effects of rosuvastatin 40 mg/day on postprandial parameters were assessed. Standardised oral fat-loading tests (OFLT) and ambulant self-measurements of daylong capillary triglycerides (TGc) were performed. Total responses of individual lipoproteins, plasma TG (TGp) and remnant-like particle cholesterol (RLP-C) were estimated as area under the curve (AUC). Most AUCs were highest in untreated patients and reached control levels after rosuvastatin. From all AUCs, RLP-C-AUC was best associated to TGp-AUC in untreated patients and controls (adjusted R^2=0.84, β=0.92, p<0.001). From all parameters of postprandial lipaemia, TGc-AUC differentiated best between untreated patients and controls (adjusted R^2=0.48, β=0.70, p<0.001) and between patients on and off-treatment (adjusted R^2=0.34, β=-0.60, p<0.001). Our findings indicate that the real-life TG load, instead of metabolic ward testing, is the best parameter of postprandial lipaemia to identify patients with premature coronary sclerosis and to evaluate postprandial effects of statin treatment.

KEYWORDS

Capillary triglycerides, lipoproteins, postprandial hyperlipidaemia, remnant-like particle cholesterol, rosuvastatin

INTRODUCTION

Fasting hypertriglyceridaemia is an independent risk factor for coronary artery disease (CAD).[1] It has been suggested that fasting plasma triglyceride (TG) concentrations are the best predictor of postprandial lipaemia.[2,3] Postprandial hyperlipidaemia is frequently present in patients with premature CAD and could therefore constitute a concealed risk factor.[4] Furthermore, exaggerated postprandial lipaemia has been observed even in fasting normolipidemic subjects.[5,6]

The usual tool to investigate postprandial lipaemia is measurement of plasma TG (TGp) and lipoprotein fraction separation during a standardised oral fat-loading test (OFLT) under metabolic ward conditions.[7,8] Recently, remnant-like particle cholesterol (RLP-C) quantification has been described to estimate the cholesterol and TG levels in atherogenic remnant lipoprotein particles.[9] Furthermore, the total TG load to which subjects are exposed during the day can be estimated by means of ambulant self-determined daylong capillary triglyceridaemia (TGc). This technique has been shown to correlate with postprandial lipaemia in the metabolic ward.[10-12]

We investigated which of the above-described methods to investigate postprandial lipaemia provides the best differentiation between patients with premature CAD before and after treatment with rosuvastatin and between those patients and matched controls.

SUBJECTS AND METHODS

Participants

The study protocol was approved by the Independent Ethics Committee of the Institutional Review Board of the

University Medical Center Utrecht and the St. Antonius Hospital Nieuwegein. Male patients aged 40 to 55 years with angiographically established CAD without any atherosclerotic event in the six months prescreening were recruited from both centres. The selection of patients was carried out by screening patients' files at random and selecting the subjects fulfilling the criteria. Exclusion criteria were diabetes mellitus, renal and/or liver disease, apolipoprotein E2/E2 genotype, body mass index >30 kg/m^2, smoking and alcohol intake >3 units/day. Fasting plasma lipids after washout of lipid-lowering medication for four weeks, fulfilled a cholesterol >5 mM and plasma TG >1.7 mM. Age- and waist-matched healthy males with fasting plasma cholesterol <6.5 mM and plasma TG <2.3 mM were recruited by advertisement. Exclusion criteria were a positive family history for premature CAD, the use of drugs known to affect lipid metabolism and the exclusion criteria used in the patients.

Study design

On each hospital visit, the participants were fasting overnight for >12 hours and did not drink alcohol on the day before. On the morning of the first visit, anthropometric measurements were performed, blood samples were drawn and the subjects received instructions for daylong TGc measurements. The second visit comprised the first OFLT and was followed by rosuvastatin 20 mg/day treatment for one month, only in the patients. Hereafter, patients visited the outpatient clinic for pill counting and control of safety parameters. Subsequently, the patients started on rosuvastatin 40 mg/day for one month, followed by a second OFLT under the same conditions as the first test. Patients self-measured daylong TGc at baseline (four weeks off treatment) and after four weeks of 40 mg/day rosuvastatin. Controls performed TGc self-measurements for one period only.

Oral fat-loading test

After inserting a venous cannula for blood sampling, subjects rested for 30 minutes before administration of the fat load. Cream was ingested within five minutes at a dose of 50 g fat and 3.75 g glucose per m^2 body surface.[13] During each test, the participants remained supine and were allowed to drink mineral water only. At regular time intervals up to ten hours postprandially blood samples were obtained in sodium EDTA (2 mg/ml) and kept on ice and centrifuged immediately for 15' at 800 g at 4°C, finally plasma was stored at -80°C.

TGc self-measurements and dietary intake

By a process of dry chemistry and colorimetry, TGc was self-measured with a TG-specific point-of-care testing device (Accutrend GCT; Roche Diagnostics, Mannheim, Germany) as described.[10-12] The measurement range for TGc is 0.80 to 6.86 mM, in the case of TGc outside this range, we used the lower or upper limit, respectively, for calculations. TGc were self-measured on three different days (preferably Monday, Wednesday and Friday; not in weekends) at six time points: fasting, pre- and exactly three hours post lunch and dinner, and at bedtime. The results were recorded in a diary, evaluated with the subjects afterwards and compared with automatically recorded data in the memory of the device. Subjects were requested to refrain from heavy physical activity; normal daily activities such as riding a bike to work were allowed. When one or more measurements were missing for a day, the data for that particular day were not used. The mean daylong TGc profile was used for statistical analysis. Results were compared with recently described cut-off levels for high (>42.5 mmol.h/l) and abnormal (between 29.5 and 42.5 mmol.h/l) daylong TGc in males.[14]

Subjects were asked to consume their usual diet, intake was unrestricted concerning the frequency and composition of the meals and was recorded in the TGc diary. Quantities of intake were estimated according to instructions provided by a dietician and by using a table with standardised portion sizes.[15] Foods consumed were converted into nutrients by using the Dutch Nutrient Database and compared with the general Dutch diet.[16]

Analytic determinations

Plasma HDL cholesterol obtained after precipitation with phosphotungstate/$MgCl_2$, and cholesterol and TG in plasma and in the isolated lipoprotein fractions were measured in duplicate by colorimetric assay with the CHOD-PAP and GPO-PAP kits respectively (Roche Diagnostics, Germany). LDL cholesterol was calculated by the Friedewald formula. Plasma apolipoprotein B (apoB) was measured by nephelometry using monoclonal antibodies (Behring Diagnostics, Germany). Plasma RLP-C isolation was based on removal of apoA-I-containing particles (HDL) and most apoB100-containing particles (LDL, nascent very-low density lipoprotein (VLDL)) by immunoseparation (Japan Immunoresearch Laboratories, Takasaki, Japan), to leave apoB48 remnants of intestinal origin and apoB100-apoE remnants of hepatic origin in the unbound fraction.[17] Cholesterol was analysed by an automated enzymatic assay (RLP-C) using a Cobas Mira S auto-analyser (ABX Diagnostics, Montpellier, France). Lipoproteins were subfractionated by discontinuous density gradient and consecutive ultracentrifugation as described.[18] The resulting lipoprotein fractions correspond with chylomicron, VLDL1 and VLDL2. Plasma insulin was measured by ELISA (Mercodia, Uppsala, Sweden). Plasma glucose, creatinine, alanine aminotransferase (ALAT), aspartate aminotransferase (ASAT) and creatinine kinase were measured by dry chemistry colorimetry (Vitros 250; Johnson & Johnson, Clinical Diagnostics, Rochester, NY, USA). ApoE

genotype was determined as described.[19] For estimation of insulin sensitivity the HOMA index (homeostasis model assessment = glucose x insulin/22.5) was calculated.

Statistics

Data are expressed as mean±SD in the text and tables and as mean±SEM in the figures. Daylong TGc was calculated as mean integrated area under the 14-hours TGc curve (TGc-AUC) and as incremental integrated area (dTGc-AUC, calculated by subtracting the baseline TGc value from following measurements) by the trapezoidal rule using GraphPad Prism version 3.0. During the ten-hour OFLTs, total and incremental AUCs were calculated as well. Differences between patients and controls were tested by Student's t-test. Effects of treatment and postprandial effects when compared with T=0h, were tested using repeated measures analysis of variance (ANOVA) with Bonferroni correction for multiple comparisons. Bivariate correlations were calculated using Spearman's correlation coefficients. Binary logistic regression analysis was performed to study which parameters differentiated best between patients and controls and between patients on and off-treatment. Linear regression analysis was performed to study predictors of postprandial lipaemia. TG, insulin and HOMA values were log transformed before analysis due to non-parametric distribution. For statistical analysis SPSS version 10.0 was used. P values <0.05 (two-tailed) were considered statistically significant.

RESULTS

Baseline characteristics

From 26 healthy subjects who responded, five were excluded (obesity: n=2, excessive use of alcohol: n=1, current smoking: n=1 and apoE2/E2 genotype: n=1). From the remaining subjects, 20 were matched for age and waist circumference with 20 CAD patients who met the inclusion criteria (*table 1*). At the start of the study, CAD patients reported a total dietary intake and relative contribution of different nutrients comparable with that of controls on a regular Dutch diet (data not shown). At the first OFLT, the untreated patients showed a less favourable fasting lipid profile and (based on HOMA) were more insulin resistant when compared with controls (*tables 1* and *2*). Baseline characteristics and fasting plasma lipids in the patients have been published elsewhere.[20] During treatment, no significant changes were observed, neither in the parameters depicted in *table 1*, nor in the self-reported dietary intake nor in physical activity (data not shown). None of the subjects started smoking. In addition, the co-medication of the patients was unchanged along the study and included aspirin (n=19), β-blockers (n=13) and angiotensin-converting enzyme (ACE) inhibitors (n=11). Rosuvastatin 40 mg/day was well tolerated and significantly improved all studied fasting lipid parameters (*table 2*).

Table 1. *Baseline characteristics of the study subjects (mean (SD))*

	Controls (n=20)	CAD patients (n=20)
Age (years)	50 (5)	50 (4)
Body mass index (kg/m²)	25.3 (2.1)	26.4 (1.4)
Waist (m)	0.92 (0.10)	0.96 (0.05)
Systolic blood pressure (mmHg)	126 (13)	129 (12)
Diastolic blood pressure (mmHg)	84 (6)	87 (8)
Glucose (mM)	5.2 (0.7)	5.2 (0.5)
Insulin (mU/l)	3.36 (3.36)	8.70 (7.19)**
HOMA index	0.77 (0.74)	2.04 (1.72)**

HOMA = homeostasis model assessment. **p<0.005.

Table 2. *Fasting plasma lipids and safety parameters of the study subjects (mean (SD))*

	Controls (n=20)	CAD patients (n=20)	
		Baseline	Rosuvastatin 40 mg/d
Plasma triglycerides (mM)	1.69 (0.59)	2.21 (0.87)*	1.52 (0.61)#
Capillary triglyceriwdes (mM)	1.29 (0.52)	3.08 (1.27)**	1.78 (0.66)##
Cholesterol (mM)	5.2 (0.9)	6.3 (1.0)**	3.6 (0.6)**##
LDL cholesterol (mM)	3.2 (0.8)	4.4 (1.0)**	1.8 (0.5)**##
RLP cholesterol (mM)	0.36 (0.11)	0.55 (0.22)**	0.33(0.11)##
HDL cholesterol (mM)	1.21 (0.25)	0.93 (0.23)**	1.10 (0.32)#
Non-HDL cholesterol (mM)	4.0 (0.9)	5.4 (0.9)**	2.5 (0.6)**##
Cholesterol/HDL cholesterol	4.4 (1.2)	7.1 (1.7)**	3.5 (0.9)**##
Apolipoprotein B (g/l)	0.97 (0.21)	1.26 (0.19)**	0.70 (0.14)**##
Creatinine (μM)	88 (7)	91 (12)	91 (12)
ASAT (U/l)	Nd	32 (10)	35 (12)
ALAT (U//)	29 (11)	34 (23)	40 (18)*
Creatinine kinase (U/l)	145 (87)	130 (91)	159 (117)

Nd = not determined; RLP = remnant-like particle. *p<0.05, **p<0.005 *vs* matched controls, #p<0.05; ##p<0.005 *vs* baseline.

Postprandial plasma TG and RLP-C

Fasting TGp, TGp-AUC and TGp-dAUC were higher in the untreated patients when compared with controls (*tables 2* and *3*, and *figure 1*). After treatment, TGp and TGp-AUC were reduced to control levels (-31%, p<0.05 and -23%, p<0.005, respectively), whereas dTGp-AUC was unaffected. In all conditions, postprandial plasma RLP-C increased towards a maximum at T=5 hours, followed by a gradual decrease towards baseline values, except for the untreated patients, where late postprandial RLP-C remained elevated (*figure 1*). Fasting plasma RLP-C and RLP-C-AUC were higher in the untreated patients when compared with controls and significantly reduced to control values by rosuvastatin (*tables 2* and *3*, and *figure 1*).

Postprandial lipoproteins

Significant postprandial changes of TG and cholesterol were not seen in IDL, LDL and HDL fractions (data not shown). In general, fasting cholesterol and cholesterol AUCs in the chylomicron, VLDL1 and VLDL2 fractions were significantly higher in the patients and reached control levels after treatment (*figure 2: left panels, table 3*). Fasting plasma TG and TG-AUC in those fractions were also higher in the patients, the reductions by treatment were small and did not reach control levels (*figure 2: right panels, table 3*).

Daylong capillary triglyceridaemia

TGc-AUC was elevated twice in the untreated patients, while dTGc-AUC was not different from controls (*figure 1*). Four controls and nine untreated patients were identified with abnormal TGc-AUC; the other patients had high TGc-AUC, the other controls normal TGc-AUC. After treatment, average TGc-AUC was significantly reduced to normal limits (<29.5 mmol.h/l), although TGc-AUC and fasting TGc remained higher when compared with controls. All treated patients showed improvement of TGc-AUC, resulting in normal, abnormal and high TGc-AUC in 12, 6 and 2 patients respectively.

Regression analyses

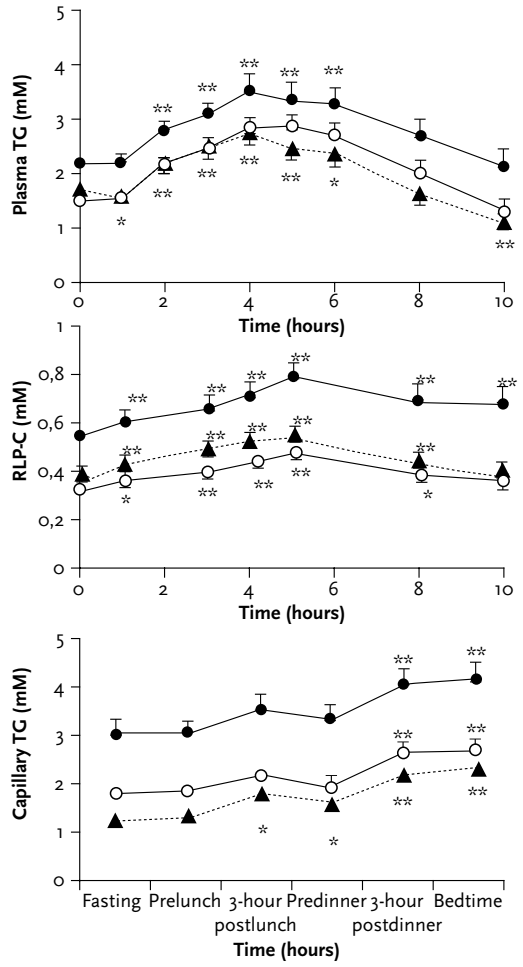
Table 4 shows that the different parameters of postprandial lipaemia (expressed as AUCs) were strongly associated. From all AUCs of *table 3*, RLP-C-AUC was the best determinant of TGp-AUC (adjusted R^2=0.84, β=0.92, p<0.001) and of TGc-AUC (adjusted R^2=0.67, β=0.82, p<0.001). When fasting lipid parameters from *tables 2*

Table 3. *Postprandial plasma lipids of the study subjects (mean (SD))*

	Controls (n=20)	CAD patients (n=20)	
		Baseline	Rosuvastatin 40 mg/d
TGp-AUC (mmol x h/l)	20.2 (8.4)	28.5 (9.2)**	22.0 (7.1)##
TGp-dAUC (mmol x h/l)	3.35 (3.83)	6.43 (4.21)*	6.83 (3.66)*
TGc-AUC (mmol x h/l)	23.6 (8.0)	47.5 (15.5)**	29.2 (8.6)*##
TGc-dAUC (mmol x h/l)	6.01 (3.62)	4.83 (6.79)	4.25 (5.16)
RLP-C-AUC (mmol x h/l)	4.69 (1.63)	6.87 (2.54)**	4.04 (1.17)##
RLP-C-dAUC (mmol x h/l)	1.06 (0.78)	1.37 (0.91)	0.75 (0.91)#
Fasting chylo-chol (mM)	0.019 (0.016)	0.027 (0.025)	0.013 (0.017)#
Chylo-chol-AUC (mmol x h/l)	0.53 (0.34)	1.16 (0.92)*	0.59 (0.26)##
Chylo-chol-dAUC (mmol x h/l)	0.34 (0.28)	0.89 (0.78)*	0.45 (0.23)#
Fasting VLDL1-chol (mM)	0.14 (0.12)	0.37 (0.21)**	0.18 (0.12)##
VLDL1-chol-AUC (mmol x h/l)	1.91 (1.32)	4.32 (1.85)**	2.54 (1.11)##
VLDL1-chol-dAUC (mmol x h/l)	0.47 (0.49)	0.61 (0.73)	0.78 (0.75)
Fasting VLDL2-chol (mM)	0.15 (0.09)	0.32 (0.16)**	0.18 (0.08)##
VLDL2-chol-AUC (mmol x h/l)	1.53 (0.91)	2.85 (1.50)**	1.64 (0.61)##
VLDL2-chol-dAUC (mmol x h/l)	0.07 (0.41)	-0.39 (0.70)	-0.11 (0.48)
Fasting chylo-TG (mM)	0.02 (0.03)	0.12 (0.17)	0.04 (0.08)
Chylo-TG-AUC (mmol x h/l)	1.95 (1.27)	4.96 (3.64)**	3.16 (1.60)*#
Chylo-TG-dAUC (mmol x h/l)	1.71 (1.08)	3.73 (3.19)*	2.77 (1.49)*
Fasting VLDL1-TG (mM)	0.36 (0.31)	0.91 (0.62)**	0.54 (0.34)*
VLDL1-TG-AUC (mmol x h/l)	4.82 (2.94)	11.15 (6.04)**	8.98 (3.84)**
VLDL1-TG-dAUC (mmol x h/l)	1.17 (1.04)	2.08 (2.45)	3.55 (2.57)**#
Fasting VLDL2-TG (mM)	0.15 (0.10)	0.36 (0.35)*	0.25 (0.14)*
VLDL2-TG-AUC (mmol x h/l)	1.72 (1.05)	3.46 (3.13)*	2.52 (1.23)*##
VLDL2-TG-dAUC (mmol x h/l)	0.274 (0.445)	-0.186 (1.603)	0.003 (0.819)

RLP = remnant-like particle; Chylo = chylomicron; chol = cholesterol; AUC = area under the curve; dAUC = incremental area under the curve, TG = triglycerides. *p<0.05; **p<0.005 *vs* age- and waist-matched controls; #p<0.05; ##p<0.005 *vs* untreated patients.

Figure 1. *Mean±SEM postprandial plasma triglycerides (TGp, upper panel) and plasma remnant-like particle cholesterol (RLP-C, middle panel) after a standardised oral fat load and self-measured diurnal capillary triglycerides (TGc, lower panel) in 20 CAD patients off lipid-lowering medication (closed bullets) and after rosuvastatin 40 mg/day (open bullets) in comparison with matched controls (n=20, dotted line)*



***p<0.05 ; **p<0.005 *vs* T=0 hours.**

and *3* were also included, TGp-AUC was best predicted by RLP-C-AUC and fasting TGp (adjusted R^2=0.86, β=0.32, p<0.001), whereas the best model to predict TGc-AUC included RLP-C-AUC and fasting TGc (adjusted R^2=0.86, β=0.32, p<0.001). When fasting TGp and TGc were excluded from this analysis (since these parameters are part of TGp-AUC and TGc-AUC), RLP-C-AUC remained the single best determinant. Logistic regression analysis with the study group (controls and untreated patients) as dependent variable and the significantly different AUCs from *table 3* as independent variables resulted in TGc-AUC as the best discriminator of patients and controls (adjusted R^2=0.48, β=0.70, p<0.001); when all significantly different parameters from *tables 2* and *3* were included, the best discriminators were fasting TGc and TGp and the total cholesterol/HDL cholesterol ratio (adjusted R^2=0.65, β=0.36, p=0.04). Similarly, from all significantly different AUCs from *table 3*, TGc-AUC was the only discriminator of treated and untreated patients (adjusted R^2=0.34, β=-0.60, p<0.001). When all significant different parameters of *tables 2* and *3* were included, non-HDL-C was the only determinant (adjusted R^2=0.77, β=-0.88, p<0.001).

DISCUSSION

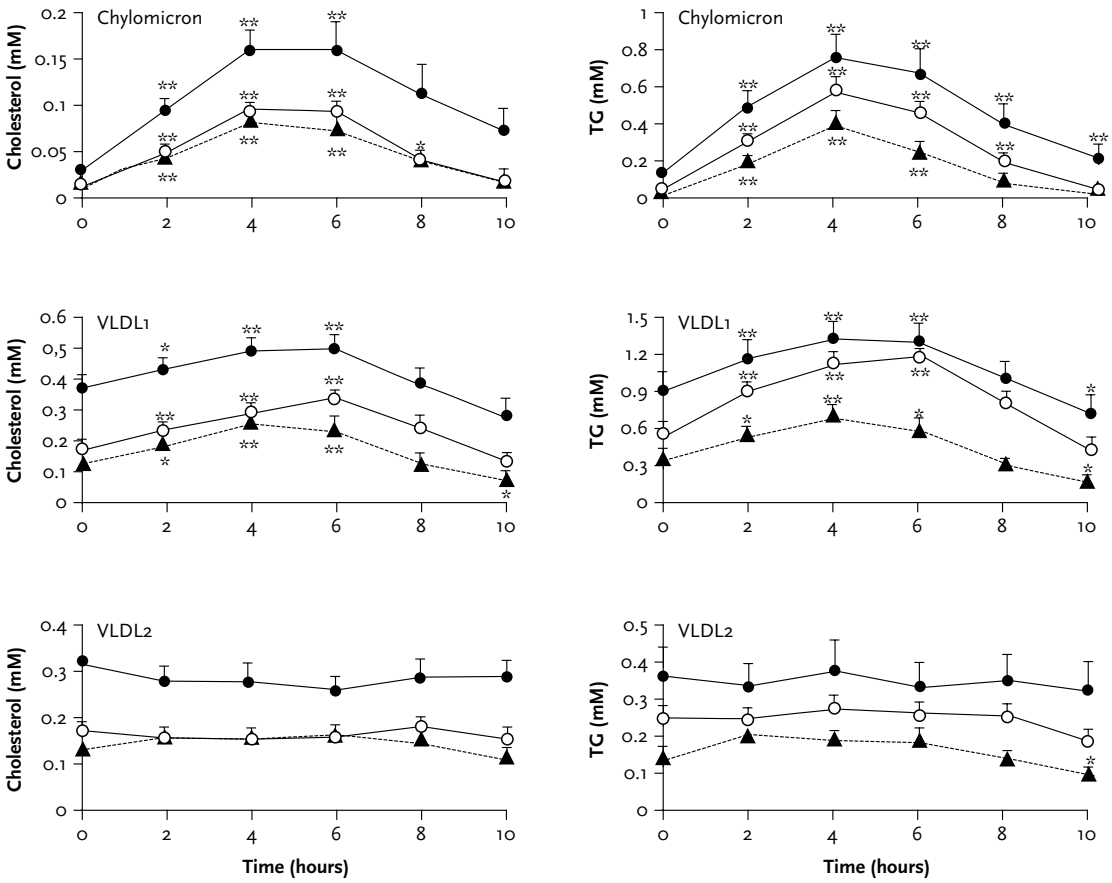
In the present study postprandial lipaemia was investigated by conventional metabolic ward testing and in a real-life situation by ambulant capillary TG self-measurement. The former makes it possible to perform lipoprotein quantification and measurement of RLP-C under controlled circumstances, since diet is known to affect lipid metabolism.[7,11,13] Usually, postprandial lipaemia is estimated by TGp-AUC. In the present study TGp-AUC showed strong associations with all other AUCs, but was best predicted by RLP-C-AUC. Furthermore, RLP-C-AUC was the best predictor of TGc-AUC. However, from all postprandial lipid parameters, the real-life TG

Table 4. *Univariate regression analysis (Spearman's correlation coefficients) with postprandial lipaemia parameters from table 3 in controls and untreated patients (n=40)*

	TGp-AUC	TGc-AUC	RLP-C-AUC	Chylo-chol-AUC	VLDL1-chol-AUC	VLDL2-chol-AUC	Chylo-TG-AUC	VLDL1-TG-AUC
TGp-AUC								
TGc-AUC	0.82							
RLP-C-AUC	0.92	0.82						
Chylo-chol-AUC	0.71	0.65	0.78					
VLDL1-chol-AUC	0.84	0.76	0.81	0.81				
VLDL2-chol-AUC	0.86	0.74	0.84	0.78	0.89			
Chylo-TG-AUC	0.69	0.68	0.72	0.83	0.85	0.74		
VLDL1-TG-AUC	0.70	0.70	0.65	0.54	0.84	0.69	0.92	
VLDL2-TG-AUC	0.70	0.66	0.73	0.62	0.79	0.85	0.83	0.91

RLP = remnant-like particle; Chylo = chylomicron; chol = cholesterol; AUC = area under the curve. For all associations p<0.001.

Figure 2. *Mean±SEM postprandial plasma cholesterol (left panels) and triglycerides (right panels) in chylomicron, VLDL1 and VLDL2 fractions after a standardised oral fat load in 20 CAD patients off lipid-lowering medication (closed bullets) and after rosuvastatin 40 mg/d (open bullets) in comparison with matched controls (n=20, dotted line)*



*p<0.05, **p<0.005 *vs* T=0 h.

load (TGc-AUC) differentiated best between untreated patients and controls and also between patients on and off-treatment. According to our data, the predictive power of TGc-AUC was better than metabolic-ward-derived parameters but also stronger than fasting lipid parameters such as LDL-C.

It was remarkable that upon treatment TGc-AUC did not reach control levels, whereas all postprandial parameters after the OFLT were reduced to levels not different from those of controls. Dietary intake, a predictor of daylong TGc,[10,11] was not different from controls and did not change during the study. A possible explanation for the discrepancy may be that daylong TGc measurements are not performed under standardised settings. For that reason, fasting TGc, the strongest predictor of TGc-AUC in the present study and in previous reports,[10] was not measured after a strict overnight fast, which may explain higher baseline TGc when compared with fasting TGp. In addition, TGc-AUC is based on averages of two or three days, which may have decreased intra-individual variability as has previously been demonstrated in healthy subjects and patients with familial combined hyperlipidaemia.[21] Finally, in some cases the detection range of TGc has caused an overestimation of TGc. In our opinion, TGc-AUC is the most realistic determination of postprandial lipaemia, since it takes into account that in real life, subjects are exposed to repetitive food intake with prolonged stressing of the lipoprotein clearance pathways. Furthermore, TGc-AUC does not exclude effects of different food components and moderate exercise on lipoprotein clearance. When compared with the OFLT, diurnal TGc is less expensive, since hospitalisation of study subjects is not necessary and laboratory techniques are cheaper; furthermore, the test is more tolerable for the subjects. We have to underline that the use of postprandial tests to identify patients at risk of CAD is still a matter of debate, since postprandial lipaemia is strongly associated to fasting lipaemia. In agreement with previous reports, we showed a strong association between fasting TGc

and diurnal TGc and also between fasting TGp and TGp-AUC.[2,3,10,22] Furthermore, when fasting TGc and TGp were included in the regression models, postprandial TG lost its power to differentiate patients from controls. Thus, fasting plasma TG identify CAD strongly. However, the aim of our study was to evaluate which postprandial variable differentiated best between CAD patients and healthy subjects.

Upon treatment with rosuvastatin all fasting lipid values were improved to levels below the latest ATP III guidelines.[23] These effects were more or less comparable with previous studies, despite the fact that our study group only showed moderate fasting hyperlipidaemia.[24,25] In line with total cholesterol, fasting and postprandial cholesterol content of individual lipoproteins was markedly reduced to levels not different from those of controls. There was a shift towards a relatively higher content of cholesterol in the HDL fraction than in the LDL fraction. By contrast, the effects of the statin on fasting and postprandial TG in lipoprotein subfractions were less pronounced. After rosuvastatin 40 mg/day, the total TGp response after a standardised oral fat load was markedly reduced to reference levels. However, of all postprandial lipaemia markers, TGc-AUC differentiated best between the pre- and post-treatment situation, again indicative of the strength of this parameter. The postprandial effects of rosuvastatin have not been reported before and are difficult to compare with other statins due to different patient groups and study meals.[26,27]

Plasma RLP-C has been shown to be a predictive marker of CAD risk and reduction of fasting RLP-C has been described upon treatment with various statins including rosuvastatin. We have shown a 40% reduction in fasting and postprandial RLP-C by rosuvastatin 40 mg/day in the present report, which is in line with two other rosuvastatin 40 mg/day studies in hyperlipidemic non-CAD patients[28] and with an atorvastatin 80 mg/day study.[29]

It is known that type 2 diabetes and the prediabetic conditions of insulin resistance and the metabolic syndrome are characterised by a disturbed TG metabolism and reduced HDL-C rather than elevated LDL-C.[30,31] The present report in subjects with reduced insulin sensitivity showed benefit of postprandial lipaemia testing, diurnal TGc in particular, when compared with determination of fasting lipid parameters. Future studies should emphasise whether other patient groups show this benefit as well.

CONCLUSION

Self-determined diurnal capillary TG seems the best and easiest method to test postprandial lipaemia to identify patients with premature CAD and to study effects of statin treatment.

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