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Postprandial lipaemia in patients with impaired fasting glucose, impaired glucose tolerance and diabetes mellitus

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ABSTRACT

Aims: To compare the postprandial lipid responses in subjects with prediabetes (IFG and IGT), newly detected diabetes mellitus (NDDM) and normal glucose tolerance (NGT).
Methods: Postprandial lipid responses to a standard oral fat challenge was studied in forty-four subjects who were divided after an OGTT into NGT, pure impaired fasting glucose (PIFG), pure impaired glucose tolerance (PIGT) and NDDM.
Results: There was a significantly higher postprandial triglyceride (PPTg) response with a higher PPTg area under curve ($p = 0.004$) and peak PPTg levels ($p = 0.003$) in patients with NDDM but not with either PIFG ($p > 0.05$) or PIGT ($p > 0.05$) when compared with NGT. Overall, PPTg responses correlated significantly with fasting plasma glucose ($p = 0.001$) and 2 h plasma glucose ($p = 0.001$) but not with age, sex, body mass index, waist, or insulin resistance.
Conclusion: Subjects with newly detected diabetes mellitus displayed postprandial hypertriglyceridemia after a standard oral fat meal challenge while no such abnormality could be demonstrated in subjects with IFG or IGT. This defect is probably related to glycemic status and insulin resistance.

1. Introduction

Macrovascular disease is a major cause of morbidity and mortality in diabetic patients [1–4]. Dyslipidemia that accompanies type 2 diabetes mellitus (DM) plays an important role in the pathogenesis of atherosclerosis in them [5–7]. Postprandial lipemia (PPL), in particular postprandial triglyceridemia is emerging as a significant independent risk factor for atherosclerosis particularly in diabetic subjects [8,9].

A strong association between postprandial triglyceridemia and early markers of atherosclerosis such as carotid intimal medial thickness (IMT) [10–13] and endothelial dysfunction [14,15] has been reported in non-diabetic as well as diabetic subjects. These and other studies support the concept that postprandial lipemia has a key role to play in the excess risk for atherosclerosis that is seen in patients with diabetes mellitus. Postprandial triglyceridemia has been shown to be consistently associated with type 2 DM in earlier studies conducted at our centre [16–18] as well as others [8,9,19].

It is well known that the atherovascular risk potential of diabetes mellitus extends to the impaired glucose tolerance (IGT) state also [20–23], although similar data with impaired fasting glucose (IFG) remains inconclusive [20,21,23]. In the light of the literature cited above it is likely that this independent excess macrovascular risk in IGT may be related to the associated proatherogenic alterations in postprandial

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lipids. There are only a few studies in literature about postprandial lipid responses in subjects with IGT [24–26] and to the best of our knowledge none in patients with IFG.

In this study we have compared the postprandial lipid responses following oral fat challenge in subjects with IFG, IGT, newly detected diabetes mellitus (NDDM) and normal glucose tolerance (NGT).

2. Materials and methods

Oral fat challenge was carried out in 44 subjects above 30 years of age of both genders who were not known diabetic patients. Those with evidence of liver or kidney disease, endocrine disease affecting lipids (hypothyroidism, Cushing's syndrome), hypertriglyceridemia, (fasting serum triglycerides (FTg) ≥250 mg/dl), recent systemic illness, inherited disorder of lipid metabolism, hypertension, congestive heart failure, smoking and those receiving drugs affecting lipid metabolism were excluded. Pregnant and lactating women and those on hormone replacement therapy (HRT) were also excluded. All premenopausal female subjects were studied in the same phase of the menstrual cycle, i.e. sixth to eighth day from the onset of menstruation.

An informed consent was obtained from each subject prior to entering the study.

Details of history and physical examination including anthropometric evaluation were recorded in a predesigned proforma. Screening investigations included hemoglobin concentration, liver and kidney function tests, serum uric acid and fasting serum lipid profile.

A standard 75 g oral glucose tolerance test (OGTT) was performed in all recruited subjects who were then divided into 4 study groups of 11 subjects each as follows: group I with NGT with fasting plasma glucose (FPG) <100 mg/dl and 2 h post-challenge plasma glucose 2 h plasma glucose (2hPG) <140 mg/dl, group II with pure impaired fasting glucose (PIFG) with FPG ≥100 but <126 mg/dl and 2hPG <140 mg/dl, group III with pure impaired glucose tolerance (PIGT) with FPG <100 mg/dl and 2hPG ≥140 but <200 mg/dl and group IV with NDDM with FPG ≥126 mg/dl and 2hPG ≥200 mg/dl. All the 4 study groups were matched for age, sex, BMI and waist measurement.

Oral fat challenge tests were performed in each of the study subjects following hospitalization as follows and as described by us previously [16–18]. After 14 h overnight fast, blood was collected for various biochemical parameters (0 h). The subjects were then given a standardized fatty meal containing 729 kcal/m^2 body surface area (BSA) and with 5.3 g protein, 24.75 g carbohydrate, 240 mg cholesterol and 65.2 g fat. This was in the form of whipped cream with sugar and fruits, which was given over 10–15 min. Blood samples were drawn at 2, 4, 6 and 8 h after the oral fat challenge. Serum was separated in all the samples by centrifuging it immediately after collection and stored at −20 °C for various biochemical estimations.

Serum lipids including total cholesterol (TC), triglycerides (Tg), high density lipoproteins (HDL), low density lipoproteins (LDL), and very low density lipoproteins (VLDL) were estimated in the 0, 2, 4, 6 and 8 h samples. In addition plasma glucose was measured in the 0 and 2 h samples and serum insulin was assayed in the 0 h sample as a measure of fasting insulinemia. Plasma glucose was analyzed by the glucose oxidase peroxidase method. Triglycerides were estimated by the lipase method, TC by cholesterol esterase/oxidase method, and HDL-C was quantitated in the supernatant after precipitation of other lipoproteins with phosphotungstate/magnesium. LDL was calculated using the friedwald formula. Serum insulin was assayed by radio immunoassay using IMMUNOTECH insulin IRMA kits. Postprandial lipid responses were compared between study groups by using one-way analysis of variance (ANOVA-*F*) test.

Table 1 – Baseline clinical, anthropometric and biochemical data in the four study groups

	Group I (NGT) Mean ± S.D. N = 11	Group II (PIFG) Mean ± S.D. N = 11	Group III (PIGT) Mean ± S.D. N = 11	Group IV (NDDM) Mean ± S.D. N = 11	*p*-Value
Age (years)	40.00 ± 5.66	38.09 ± 5.84	44.73 ± 9.1	40.64 ± 6.67	NS
Male/female	4/11	3/11	3/11	3/11	–
Family history +ve	5/11	2/11	3/11	3/11	–
BMI (kg/m^2)	25.07 ± 4.26	26.74 ± 5.06	28.13 ± 6.23	25.96 ± 3.17	NS
Waist (cm)	85.35 ± 9.73	92.09 ± 12.23	89.91 ± 10.35	86.78 ± 7.57	NS
Waist:hip	0.94 ± 0.05	0.92 ± 0.04	0.91 ± 0.10	0.93 ± 0.05	NS
FPG (mg/dl)	89.55 ± 5.34	107.36 ± 6.04	90.91 ± 6.47	211.45 ± 68.05	A, 0.000; B, 0.000; C, 0.000
2hPG (mg/dl)	107.00 ± 13.67	128.45 ± 8.72	157.73 ± 11.06	347.18 ± 99.48	A, 0.000; B, 0.000; C, 0.000
Fasting serum insulin (mu/l)	6.22 ± 4.54	9.57 ± 6.24	11.68 ± 8.86	7.99 ± 5.04	NS
Total cholesterol (mg/dl)	160.27 ± 51.26	176 ± 31.49	181.09 ± 21.19	174 ± 31.71	NS
Triglyceride (mg/dl)	92.91 ± 45.75	137.45 ± 36.79	125.64 ± 29.19	186.73 ± 89.09	A, 0.005
HDL (mg/dl)	44.73 ± 9.19	42.45 ± 11.38	41.45 ± 7.31	34.91 ± 8.96	NS
LDL (mg/dl)	97 ± 49.93	107.36 ± 29.70	114.64 ± 15.83	102 ± 24.47	NS
VLDL (mg/dl)	18.55 ± 8.94	27.09 ± 7.65	25 ± 5.78	37.09 ± 17.79	A, 0.006

A, group I vs. group IV; B, group II vs. group IV; C, group III vs. group IV; NGT, normal glucose tolerance; PIFG, pure impaired fasting glucose; PIGT, pure impaired glucose tolerance; NDDM, newly detected diabetes mellitus; BMI, body mass index; FPG, fasting plasma glucose; 2hPG, 2 h plasma glucose; HDL, high density lipoprotein; LDL, low density lipoprotein; VLDL, very low density lipoprotein.