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Glucose tolerance and lipid profile in survivors of myocardial infarction

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Objective In order to reduce the mortality and morbidity from coronary heart disease, further risk factors are evaluated.

Method Fortysix patients surviving myocardial infarction without diabetes and 25 control subjects were subjected to oral glucose tolerance test, and their glucose and lipid profiles were compared.

Result Patient group showed significantly increased plasma glucose levels at the 30th, 60th, 90th and 120th minutes ($p<0.001$, $p<0.01$, $p<0.05$ and $p<0.05$, respectively) compared with the control group. HbA_{1c} and fructosamine levels were significantly higher in the patient group ($p<0.01$). Abnormal glucose tolerance was observed in 9 (19.5%) patients. One of these showed diabetic curve. Impaired glucose tolerance (IGT) was observed in 1 control subject. Serum cholesterol, triglyceride and low-density lipoprotein cholesterol values were also higher in the patient group ($p<0.001$, $p<0.01$, $p<0.001$, respectively). Serum high-density lipoprotein cholesterol value was lower in the patient group ($p<0.001$).

Conclusion These results suggest that IGT and related conditions are important risk factors for coronary heart disease, and subjects with glucose intolerance and lipid disorders should be early detected and taken to treatment program.

Key words Myocardial infarction, glucose tolerance test, lipids

Introduction

In the last 30 years, considerable advances have been achieved in the determination and improvement of risk factors, such as cigarette smoking, high blood pressure and high serum total and low-density lipoprotein (LDL) cholesterol levels, for cardiovascular disease (CVD). These advances have caused an important decline in the incidence of CVD in some countries. However, despite this positive result, CVD is still among the first cases of death in most Western countries. Further decline in cardiovascular morbidity and mortality might be achieved if further risk factors can be found (1).

Although diabetes mellitus is usually recognized as a major risk factor for coronary heart disease (CHD) morbidity and mortality, in several studies it has not been found responsible as a single factor (2,3). Recently, it has been suggested that Impaired glucose tolerance (IGT) may also become an independent risk factor (4). Even Helsinki Policemen Study has shown that subjects whose blood glucose lies in the upper 20% of the population have increased risk of subsequent CHD (5) .

We investigated glucose levels and frequency of IGT in survivors of MI, comparing with control subjects to last the study in earlier time, because the observation of development of CHD in subjects with IGT required long-term follow-up.

Material and Method

The present study comprised 46 patients between 35 and 69 years of age who had MI diagnosed at least established if at least two of the following three criteria were fulfilled: characteristic clinical symptoms with chest pain for at least 20 minutes; development of abnormal Q waves or permanent ST-T changes; transient increase in myocardial enzymes. All patients were carefully screened to exclude evidence of congestive heart failure, hepatic and renal failure, endocrinological disorders and other ailment or drug intake known to affect glucose tolerance. Subjects who had fasting plasma glucose of ≥ 140 mg/dl were excluded. Twenty five age and sex matched healthy controls were also selected. All subjects were physically active and had an unrestricted diet (150g/day of carbohydrate) for the last 3 days before the glucose tolerance test (GTT). After an overnight 12-h fasting, blood samples were taken for measurement of glucose, total cholesterol, high-density lipoprotein (HDL) cholesterol, LDL cholesterol, triglyceride, glycosylated hemoglobin (HbA_{1c}), and fructosamine, followed by a 2-h 75-g oral GTT. During GTT, blood samples were taken at every 30 mins. interval for 2 hours. Glucose tolerance was classified using the WHO criteria. Body mass index (BMI) was calculated as weight in kilograms divided by height in meter square (kg/m2). Blood pressure was measured with a standard sphyngomanometer and after 10 mins. of rest. Two BP measurements were made in the supine position and the mean was recorded.

Plasma glucose, cholesterol, triglyceride and serum HDL cholesterol concentrations were assayed by commercial enzymatic methods. Serum LDL cholesterol concentration was calculated with Friedwald equation in patients with triglyceridemia

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<400 mg/dl (6). Serum fructosamine level was determined by a commercial spectrophotometric method. HbA_{1c} level was determined by quantitative colorimetric determination.

Data analyses were performed with the SPSS for Windows program (Statistical Package for Social Sciences). Results were expressed as mean ± SD. Differences between group means were compared using the Student's unpaired *t* test.

Results

Clinical characteristics of the groups are shown in table I. Patient group showed a significant increase in plasma glucose values at the 30th ($p<0.001$), 60th ($p<0.01$), the 90th ($p<0.05$) and 120th min. ($p<0.05$) compared with control group. Fasting plasma glucose values in both groups were statistically insignificant ($p>0.05$). HbA_{1c} and fructosamine levels were significantly higher in the patient group ($p<0.01$). Abnormal glucose tolerance was observed in 9 (19.5%) patients. One of these was a diabetic curve. IGT was observed in 1 control subject. Serum cholesterol, triglyceride and LDL cholesterol values were also higher in the patient group ($p<0.001$, $p<0.01$, $p<0.001$, respectively). However serum HDL cholesterol value was lower ($p<0.001$). Results are shown in Table II. Both systolic and diastolic blood pressures were higher in the patient group ($p<0.01$). Smoking was more common in men in both groups but there were more subjects (59%) in the patient group (before MI), compared with the control group (36%).

Discussion

The body responds to stress, e.g., during infections or acute illnesses, by a variety of neurohormonal mechanisms. Some of these physiological responses sometimes lead to an elevation of the plasma glucose and triglyceride levels. Conversely, plasma total cholesterol and HDL cholesterol may be diminished, and the fall in HDL cholesterol may persist for 6 to 8 weeks in myocardial infarction (7-9). Therefore, we performed OGTT, and measured lipid parameters in 46 patients at least 3 months after the infarction.

In this study we have confirmed the high incidence of IGT in patients with MI. In many studies (e.g. Honolulu Heart Study, Whitehall Civil Servants Study, Tecumseh Study, and Chicago Gas Company Investigations), the hyperglycemia during OGTT has been considered an independent risk factor in the development of CHD (10,11). But some other studies, including Paris Prospective Study, have shown that neither diabetes nor IGT were significant independent risk factors for CHD death (2,12).

Table I. Clinical characteristics of the groups

	Control subjects (n=25)	Patients (n=46)	*p* value
Sex (M/F)	16/9	30/16	
Age (years)	53 ± 7	54 ± 8	
BMI (kg/m^2)	27.2 ± 2.8	27.3 ± 3.4	
sBP (mmHg)	137 ± 18.5	149 ± 17.5	<0.01
dBP (mmHg)	84 ± 12.2	92 ± 9.1	<0.01
Smokers n (%)	9 (36)	27 (59)	
Smokers M/F	7/2	24/3	

The data are mean ± SD.

Table II. Glucose and lipid profiles in patients and control subjects

	Control subjects	Patients	*p* value
Plasma glucose (mg/dl)			
Fasting	88.1 ±10.8	92.6 ± 14.5	> 0.05
30 min	126.6 ± 19.3	152.4 ± 33.3	< 0.001
60 min	137.0 ± 22.8	163.5 ± 51.7	< 0.01
90 min	112.7 ± 21.1	133.2 ± 49.7	< 0.05
120 min	95.4 ± 14.3	109.8 ± 41.6	< 0.05
HbA_{1c} (%)	4.5 ± 0.4	4.9 ± 0.7	< 0.01
Fructosamine(mmol/l)	1.62 ± 0.2	1.77 ± 0.2	< 0.01
Serum cholesterol (mg/dl)	194.3 ± 30.3	233.8 ± 37.4	< 0.001
Serum triglyceride (mg/dl)	131.3 ± 50.2	173.5 ± 66.9	< 0.01
HDL cholesterol (mg/dl)	46.8 ± 6.9	38.2 ± 7.9	< 0.001
LDL cholesterol (mg/dl)	121.2 ± 25.8	161.3 ± 35.4	< 0.001

The data are mean ± SD.

However, The Paris Prospective study demonstrated that compared with normoglycemic men the relative risk of death in subjects with IGT was 1.6 (13).

In contrast, The Framingham Study has suggested that hyperglycemia is an independent risk factor for CVD in nondiabetic women, but not among men (11).

IGT is more frequently found in survivors of myocardial infarction (4), although Peters and Hales reported lower mean blood glucose in patients of MI (14). Gupta et al observed abnormal glucose tolerance in 32% of patients with MI (15). We observed it in 19.5% of patients. This distinction might be caused from the selection of patients (we excluded subjects with known diabetes and had fasting plasma glucose ≥140mg/dl at baseline).

The OGTT is widely used for the diagnosis of diabetes and impaired glucose tolerance. Determination of glycosylated hemoglobin and total serum proteins (fructosamine) has been proposed as an alternative method of screening. Both glycosylated hemoglobin and fructosamine have been found to have good specificity (100 and 97%, respectively), but low sensitivity (15 and 19%, respectively) (16). In this study, both HbA1c and fructosamine levels were higher in the patient group, compared with the control ($p<0.01$).

Kannel and Wilson reported that LDL cholesterol levels predicted CHD only marginally better than serum total cholesterol. However triglyceride levels were the weakest predictor, but HDL cholesterol was the best single lipid predictor. The use of HDL cholesterol avoided needlessly alarming or falsely reassuring persons at risk for CHD who had high total cholesterol values (17). These findings approximately concur with our results which show statistically more significant values for total cholesterol, HDL and LDL cholesterol levels ($p<0.001$) than triglyceride levels ($p<0.01$) .

Based on these findings, subjects with lipid abnormalities or glucose intolerance should be early detected and carefully managed to prevent MI, because subjects with MI have more plasma lipid abnormalities and increased glucose levels.

References

1. Assmann G, Schulte H, Eckardstein A. Hipertrigliseridemi/ düşük HDL kolesterolü sendromu. Born GV, Schwartz CJ, eds. Koroner Kalp Hastalığında Yeni Ufuklar: İstanbul: Turgut Yayıncılık ve Ticaret, 61-71, 1995.
2. Eschwege E, Balkau B, Fontbonne A. The epidemiology of coronary heart disease in glucose-intolerant and diabetic subjects. J Intern Med Suppl 736:5-11, 1994.
3. Yudkin JS. Coronary heart disease in diabetes mellitus: three new risk factors and a unifying hypothesis. J Intern Med 238:21-30, 1995.
4. Yamamoto A, Yamamura T, Kawaguchi A. Triglyceride and glucose intolerance as a risk factor for coronary heart disease. Cardiology 78:185-193, 1991.
5. Swales J, Bono D: Cardiovascular Risk Factors, London: Times Mirror International Publishers Limited, 88-97, 1994.
6. Ganda OP. Disorders of lipid metabolism. Lavin N, eds. Manual of Endocrinology and Metabolism: Boston: Little, Brown and Company, 477-491, 1994.
7. Mak KH, Mah PK, Tey BH, Sin FL, Chia G. Fasting blood sugar level: a determinant for in-hospital outcome in patients with first myocardial infarction and without glucose intolerance. Annals of the Academy of Medicine 22:291-295, 1993.
8. Madsen JK, Haunsoe S, Helquist S, Hommel E, Malthe I, Pedersen NT, Sengelov H, Jessen DR, Telmer S, Parving HH. Prevalence of hyperglycaemia and undiagnosed diabetes mellitus in patients with acute myocardial infarction. Acta Med Scand 220: 329-332, 1986.
9. Sobel BE. Acute myocardial infarction. In: Wyngaarden JB, Smith LH, Bennett JC, eds. Cecil Textbook of Medicine: Philadelphia: WB Saunders Company, 304-318, 1992.
10. Butler WJ, Ostrander LD , Carman WJ, Lamphiear DE. Mortality from coronary heart disease in the Tecumseh study. Long-term effect of diabetes mellitus, glucose tolerance and other risk factors. Am J Epidemiol 121:541-547, 1985.
11. Wilson PW, Cupples LA, Kannel WB. Is hyperglycemia associated with cardiovascular disease? The Framingham Study. Am Heart J 121:586-590, 1991.
12. Fontbonne AM, Eschwege EM. Insulin and cardiovascular disease. Paris Prospective Study. Diabetes Care 14:461-469, 1991.
13. Balkau B, Eschwege E, Papoz L, Richard JL, Claude JR, Warnet JM, Ducimetiere P. Risk factors for early death in non-insulin dependent diabetes and men with known glucose tolerance status. BMJ 307:295-299, 1993.
14. Peters H, Hales CN. Plasma insulin concentrations after myocardial infarction. Lancet 1: 1144-1145, 1965.
15. Gupta MS, Gulati JK, Malhotra KC. Insulin response and glucose tolerance in survivors of myocardial infarction. JAPI 37:255-257, 1989.
16. Guillausseau PJ, Charles MA, Paolaggi F, Timsit J, Chanson P, Peynet J, Godard V, Eschwege E, Rousselet F, Lubetzki J. Comparison of HbA1 and fructosamine in diagnosis of glucose-tolerance abnormalities. Diabetes Care 13:898-900, 1990.
17. Kannel WB, Wilson PW. Efficacy of lipid profiles in prediction of coronary disease. Am Heart J 124:768-774, 1992.

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