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Renal function and haemodynamics in obese hypertensive patients

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Abdominal obesity and hypertension being linked via insulin resistance lead to kidney impairment. It is of practical interest to estimate the contribution of each factor in the renal circulation and function lesions. The impact of obesity on renal haemodynamic and glomerular filtration of hypertensive subjects was investigated.

17 lean and 30 stout patients with mild to moderate hypertension and normal oral glucose tolerance (OGTT) test were investigated. 19 subjects of the obese group have central fat distribution. The plasma levels of insulin (fasting and during OGTT) and cortisol were assessed by means of radioimmuno assay. The effective renal blood and plasma flow (RBF and RPF) and glomerular filtration rate (GFR) were determined by radionuclide method. RPF and GFR expressed as absolute values and related to the height (but not to the body surface area of 1.73 m²) were higher in hypertensive subjects with abdominal obesity (RPF 492.1 ± 28.3 vs. 418.6 ± 22.3 ml/min, *p* = 0.02 in lean hypertensives and RPF/height 2.85 ± 0.16 vs. 2.42 ± 0.13 ml/min per cm, *p* = 0.016, GFR 132.3 ± 9.6 vs. 96.8 ± 12.3 ml/min in lean subjects, *p* = 0.01 and GFR/height 0.77 ± 0.05 vs. 0.54 ± 0.07 ml/min per cm, *p* = 0.01, resp.)

Abdominal obesity in hypertensive patients is associated with renal vasodilatation and glomerular hyperfiltration which are not caused by direct influence of hyperinsulinemia. *J Clin Basic Cardiol* 1999; 2: 73–4.

Key words: Obesity, hypertension, insulin, kidney, regional blood flow

Hypertension has serious impact on renal circulation and function. Insulin resistant states seem to promote intraglomerular hypertension [1]. An increased rate of urinary albumin excretion was demonstrated for non-insulin-dependent diabetes mellitus [2]. In case of obesity the situation is not so obvious, possibly because of heterogeneity of this metabolic disorder. The influence of obesity on the kidney of a hypertensive subject is poorly elucidated. Microalbuminuria is considered to be the universally used screening method for the glomerular vascular permeability assessment. Otherwise, direct and simultaneous estimation of glomerular filtration rate and renal plasma flow by means of the radionuclide method is a rather precise tool for regional pathogenic mechanism investigation [3]. The present study was undertaken in order to estimate the effect of obesity on renal function and haemodynamics in hypertensive subjects.

Methods

We examined 47 hypertensive patients (26 males and 21 females) aged 34–56 years with mild-to-moderate hypertension. 30 of them were obese subjects. Obesity and central fat distribution were defined, respectively according to Body Mass Index (BMI > 27 kg/m²) and waist to hip ratio (WHR). 19 fat patients had central type of obesity (WHR > 0.95 for men and > 0.83 for women). The second group consisted of 17 normal weight hypertensive individuals (NWHT) who were sex, age and blood pressure (BP) level matched with the patients of the total group of obesity and subgroup of central obesity.

Oral glucose tolerance test was performed according to WHO recommendations [4]. Blood samples were drawn at baseline and every 30 min for 2 hours after ingestion of 75 g glucose to measure plasma glucose concentration. Serum insulin was determined by a standard radioimmune method (solid phase I-125 radioimmune assay). The values of glucose and insulin concentrations obtained during OGTT have been expressed as area under the curve measured using the trapezoidal

rule. Basal levels of serum cortisol and beta-2-microglobulin (B2MG) also were assessed by means of radioimmuno assay. The effective renal blood and plasma flow and glomerular filtration rate were determined by single injection of Tc-99m-Pentatech and I-131-hippurate, renal vascular resistance (RVR), and filtration fraction of RPF being calculated. All measurements of renal function and haemodynamics were given as absolute values and after correction to 1.73 m² body surface area (BSA).

All statistical calculations were carried out using the statistical package Statgraphics. The results are expressed as mean ± standard error of mean (SEM). All variables were normally distributed and the variances were homogeneous across the groups. Statistical difference was performed using the Student's *t*-test for unpaired data. Correlations were performed using linear regression analysis.

The study was approved by an institute review committee and the subjects gave informed consent.

Results

The levels of fasting insulin as well as the area under the insulin curve during the OGTT were higher in hypertensive subjects with abdominal obesity (Table 1).

The renal plasma flow related to body surface area (BSA) of 1.73 m² was similar in lean and stout subjects, while expressed as absolute values, it was significantly higher in patients with abdominal obesity. The level of renal vascular resistance was 20.4 % lower in patients with central obesity vs. lean hypertensive subjects (*p* = 0.002).

RPF correlated with the duration of hypertension in NWHT patients (*r* = 0.66, *p* = 0.001) while no relation between these variables was mentioned in obese individuals. GFR (expressed as absolute values) was increased in centrally obese compared with lean subjects. The higher the levels of insulin and systolic BP in obese patients (total group), the lower was the GFR (*r* = 0.50, *p* = 0.03 and *r* = 0.56, *p* = 0.007 respectively). The filtration fraction of RPF also was higher in stout patients though the difference with to subjects was significant

Table 1. Haemodynamic and humoral parameters of lean and stout patients with essential hypertension

	Lean hypertensive subjects	Obese hypertensive subjects		
	n = 17	All n = 30	Central type of obesity n = 19	Periph. type of obesity n = 11
Men/Women	10/7	16/14	12/7	4/7
Age, years	43.6 ± 2.6	42.8 ± 2.1	44.1 ± 1.8	40.1 ± 3.2
History of Hypertension, yrs	7.6 ± 1.6	8.3 ± 1.9	7.6 ± 2.0	10.1 ± 4.8
Height, cm	172.6 ± 1.3	168.7 ± 1.5	172.3 ± 1.9	164.0 ± 2.4*
BMI, kg/m ²	23.5 ± 0.4	35.2 ± 0.8*	35.7 ± 1.0*	34.9 ± 1.3*
BPs, mmHg	136.5 ± 3.76	144.3 ± 3.7	146.4 ± 5.0	141.2 ± 6.8
BPd, mmHg	89.6 ± 1.9	92.9 ± 1	96.5 ± 2.5	88.1 ± 2.5
RPF, ml/min 1.73 m ²	396.9 ± 23.0	382.6 ± 18.9	399.0 ± 23.9	323.2 ± 46.4
ml/min	418.6 ± 22.3	469.0 ± 24.0	492.1 ± 28.3*	420.2 ± 36.6
ml/min per cm	2.42 ± 0.13	2.75 ± 0.13	2.85 ± 0.16*	2.34 ± 0.38
RVR, dyns x s/cm ⁵	11,677 ± 950.6	9462.9 ± 230.5	9298.4 ± 281.7*	9670 ± 260.0
GFR, ml/min 1.73 m ²	92.1 ± 11.6	105.3 ± 5.4	108.2 ± 6.0	98.0 ± 4.6
ml/min	96.8 ± 12.3	129.8 ± 7.6	132.3 ± 9.6*	107.2 ± 10.4
ml/min per cm	0.54 ± 0.07	0.74 ± 0.04	0.77 ± 0.05*	0.70 ± 0.15
Filtr. fraction of RPF, %	23.7 ± 3.71	27.2 ± 2.0	28.2 ± 2.3*	26.0 ± 2.5
B2MG, mg/L	1.8 ± 0.2	2.42 ± 0.3	2.37 ± 0.37	2.51 ± 0.78
Fasting glucose, mmol/L	4.4 ± 0.3	4.5 ± 0.2	4.7 ± 0.4	4.4 ± 0.2
Glucose area, mmol* min/L	783.1 ± 42.2	814.0 ± 31.3	816.5 ± 30.3	810.4 ± 41.8
Fasting insulin, mU/L	10.2 ± 2.6	16.8 ± 2.0	17.5 ± 3.0*	16.4 ± 3.3
Insulin area, mU*min/L	7224.6 ± 230.2	8031.2 ± 210.8*	8027.5 ± 203.0*	8046.8 ± 309.1*
Cortisol, nmol/L	404.3 ± 88.6	467.0 ± 77.6	430.1 ± 94.1	504.3 ± 143.9

* = p < 0.05 vs lean hypertensive subjects

only for central obesity subgroup. The levels of B2MG in obese and NWHT groups did not differ significantly. The expected negative correlation between the levels of B2MG and GFR was revealed only in hypertensive subjects with normal BMI ($r = 0.49$, $p = 0.0004$).

Discussion

These results indicate that central obesity in hypertensive subjects is associated with renal vasodilatation and glomerular hyperfiltration. This phenomenon is not caused by direct influence of hyperinsulinaemia. Our results can be matched with those obtained with microalbuminuria assessment [5].

It should be mentioned that the difference between lean and central obese subjects was revealed when we analyzed renal haemodynamic and function parameters expressed as absolute values rather than indexed to ideal body surface area. We are in agreement with critical remarks that traditional indexing to 1.73 m² in the case of stout subjects leads to unjustified ("arithmetical" but not real) lowering of the kidney circulation parameters. Body surface area as well as body mass index were not independent predictors of renal blood and plasma flows while height was as reported by Schmieder et al. [6]. When we indexed RPF and GFR values to the height the difference between lean subjects and hypertensive patients with central obesity remained significant ($p = 0.02$).

The revealed hyperfiltration may be ascribed to kidney autoregulation impairment and additional afferent vasodilatation. Such influence of obesity on renal circulation was previously reported [7, 8]. The second possible reason for hyperfiltration

in stout patients is intrarenal hypertension due to renal interstitial cell proliferation and matrix amount enlargement [9, 10]. As was postulated by Hall 1997, the increase of the glomerular filtration rate in these conditions is the way to counteract increased loop reabsorption and to avoid sodium retention [11].

Thus, abdominal obesity modifies the haemodynamical influences of hypertension on circulation and function of the kidney and additional renal impairment must be anticipated.

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