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Serum Lipoprotein(a) Is Not Increased in NIDDM Patients With Microalbuminuria

Recently, much interest has been focused on lipoprotein(a) (Lp[a]) and diabetes (1). Particularly, contrasting data are available in the literature about the levels of Lp(a) in non-insulin-dependent diabetes mellitus (NIDDM) and its possible relationships with incipient diabetic nephropathy, i.e., with microalbuminuria (2–4). We determined Lp(a) levels and lipoprotein patterns in Caucasian NIDDM patients grouped according to their urinary albumin excretion (UAE) rate determined on three consecutive 24-h urine samples. Of the patients, 69 were microalbuminuric (UAE rate between 30 and 300 mg/24 h, median value and range: 66 [30–281] mg/24 h) and 60 were normoalbuminuric (UAE rate < 30 mg/24 h, 8.2 [5.0–27] mg/24 h). Sixty healthy individuals served as control subjects. The three groups of subjects were comparable for sex, age, duration of diabetes, prevalence of hypertension (47 vs. 61%), cardiopathy (26 vs. 30%), arteriopathy (14 vs. 14%), cardiovascular accidents (13 vs. 14%), retinopathy (24 vs. 18%), and glycometabolic control (Table 1). None of the diabetic patients or control subjects was affected by diseases or taking any medications known to influence Lp(a) levels. All diabetic patients were on diet and/or hypoglycemic agents.

Lp(a) serum concentrations were measured by a commercial ELISA sandwich method, using a polyclonal antibody (Biopool, Umea, Sweden) (interassay and intrassay coefficient of variation were 5 and 12%, respectively). The Mann-Whitney test was used to compare distributions. Correlations between parameters were looked at with the Kendall correlation test.

The median concentration of Lp(a) in microalbuminuric patients (9.2 mg/dl, range 0.1–116.8) was not significantly different with respect to that found in patients with normal UAE rates (8.0 mg/dl, range 0.3–69) (Table 1). Furthermore, the median Lp(a) concentration was not significantly different in the whole group of NIDDM patients (8.2 mg/dl, range 0.1–116.8) with respect to control subjects (9.2 mg/dl, range 1.6–64.5). We did not find any positive correlation between Lp(a) serum concentration and UAE rate in either normo- or microalbuminuric NIDDM patients or in the whole population of diabetic subjects. In both groups of diabetic patients, the mean HbA_{1c} and blood glucose levels did not show a positive correlation with serum Lp(a) concentrations in both the normoalbuminuric and microalbuminuric patients. Median concentrations of serum triglycerides and apolipoprotein B were significantly higher in microalbuminuric with respect to normoalbuminuric patients ($P < 0.001$ and $P < 0.05$, respectively), whereas the two groups did not show any significant difference in the median of other lipidic parameters.

Our findings are in contrast with those reported previously by Jenkins et al. (2): apolipoprotein A serum concentration in a relatively small sample of microalbuminuric NIDDM patients ($n = 26$) was higher than in normoalbuminuric subjects ($n = 56$). However, we included more than twice as many patients with an abnormal UAE rate compared with the previous study. Based on our data and taking into consideration the well-described ethnic influences on Lp(a) (5), it cannot be excluded that the discrepancies in the results between the two studies could be caused by genetic and/or racial differences of the populations studied. Another possible factor confounding interpretation of the results of Jenkins et al.'s (6) study may be the investigation of insulin-treated NIDDM.

In conclusion, we confirm another study (7) showing that NIDDM subjects do not have higher Lp(a) levels with respect to the general population. The main finding of our study is that Lp(a) levels were not associated with microalbuminuria in NIDDM patients treated with diet and hypoglycemic agents with relatively good glycometabolic control. Among NIDDM patients with normal UAE rates and with microalbuminuria, 16 and 17 subjects, respectively, had a serum Lp(a) concentration higher than 25 mg/dl, which is the usual cutoff value identifying subjects at high cardiovascular risk. Interestingly, in the microalbuminuric patients, the median Lp(a) concentration above this cut off was significantly higher than for normoalbuminuric patients (52 mg/dl, range 28.2–116.8 mg/dl vs. 32.7 mg/dl, range 26.2–69 mg/dl; $P < 0.05$). It could be hypothesized that only these latter patients may constitute the subgroup of diabetic

Table 1—*Clinical and metabolic characteristics of NIDDM patients with and without microalbuminuria*

	Diabetic patients		
	Normoalbuminuric	Microalbuminuric	*P* value
n	70	69	
Sex (M/F)	38/32	40/29	NS
Age (years)	56 (39–68)	58 (35–67)	NS
Duration of diabetes (years)	8.0 (1–27)	8.0 (1–26)	NS
HbA_{1c} (%)	7.2 (4.6–12.1)	7.1 (4.4–12.4)	NS
Lp(a) (mg/dl)	8.0 (0.3–69)	9.2 (0.1–116.8)	NS
Cholesterol (mg/dl)	219.5 (125–329)	232 (130–347)	NS
Triglycerides (mg/dl)	124.5 (28–739)	184.0 (60–533)	<0.001
High-density lipoprotein cholesterol (mg/dl)	38 (17–99)	36 (18–73)	NS
Low-density lipoprotein cholesterol (mg/dl)	151.7 (59.6–257)	153.4 (76–257)	NS
Apolipoprotein A (mg/dl)	133.5 (71–189)	129 (95.6–222)	NS
Apolipoprotein B (mg/dl)	124.5 (56.8–333)	142 (92.8–240)	0.025

Data are median (range).

patients likely to develop macrovascular complications.

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Pancreatic Metastases of Grawitz' Tumor Revealed by Ketoacidosis

Pancreatic metastases of renal cell carcinoma are a rare occurrence, expected in 1–3% of patients who develop a metastatic disease (1). Impaired carbohydrate metabolism is a common feature in chronic pancreatic diseases, but multiple metastases of the pancreas from a renal cell carcinoma revealed by diabetic ketoacidosis are exceptional. Only one case has been reported (2). Here we report a new observation in which ketoacidosis was the first sign of the recurrence of a renal cancer.

In 1991, a 55-year-old man was referred to our institute for an inaugural diabetic ketoacidosis. He had no history of diabetes. He had a surgical pituitary insufficiency (substituted by hydrocortisone [30 mg/day] and L-thyroxine [125 μg/day]) after the removal of a craniopharyngioma in 1978. In 1987, a left nephrectomy was performed for a renal cell carcinoma. The tumor was confined within the perirenal fascia (stage T2, N0, M0), and no local recurrence was found in the postoperative follow-up. On admission, the patient complained of asthenia, nausea, polyuropolydipsic syndrome with dehydration, and moderate weight loss. The palpation of his abdomen was normal and painless. Blood pressure was 125/75 mmHg, and body temperature was 37.3°C. Clinical examination and electrocardiogram found no other abnor-