Red Cell, Plasma and Urine Amino Acid Concentrations in Patient with Diabetes Mellitus Type I.

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The concentrations of amino acids (AA) in blood plasma, urine and erythrocytes were determined in patients with diabetes mellitus (DM) type I and a control group. Plasma concentrations of Aba, Asp, Cit, His, Ile, Leu, Orn, Phe, Tyr were significantly increased in DM I, while most AA in erythrocytes were decreased except Arg, Asp, and Tyr. In urine higher losses of AA were recorded in the group of patients. These findings confirm that in patients with DM I intracellular metabolism of AA is disturbed.

INTRODUCTION

Insulin deficiency in patients with DM reveals by increased protein catabolism followed hyperaminoacidemia. Insulin inhibits proteolysis and so increases protein anabolism [2]. The effect of exogenous insulin on lysine transport system was described [4]. During sepsis insulin resistance was observed and the effect of insuline on AA transport was decreased [3].In septic rats levels of neutral AA were mainly changed and insulin administration together with AA did not show any effect. Disturbances of AA metabolism in plasma, erythrocytes and muscle of patients with uremia were depicted [1].

The aim of our work was to evaluate AA levels in plasma and erythrocytes depending on excretion by urine in patients with DM I treated by insulin.

MATERIALS AND METHODS

The levels of AA in plasma, urine and erythrocytes were examined in 33 patients with DM I with the average age 39.4 ± 13.8 years by amino acid analyser AAA 339-T (Mikrotechna Prague). The mean lasting of diabetes was 13.0 ± 9.7 years.The concentrations of plasma AA were expressed in μmol/l, of urine AA were recalculated to μmol/24 h, and of erythrocyte AA to μmol/g Hb. The results were compared with a control healthy group of approximate size (n = 35) and age by nonpaired Student t-test.

Table 1. Statistically significant correlation between DM diagnostic parameters and amino acid levels in red cells, urine and blood plasma.

Parameters		*N*	r	*P* <
RED CELLS				
GHb	ALA	32	+0.315	0.05
	GLU	32	−0.325	0.05
	LYS	31	+0.340	0.05
	TRP	29	+0.432	0.01
URINE (24 h)				
Glycaemia	ABA	26	+0.523	0.01
	LEU	29	+0.328	0.05
Glycosuri	ALA	28	+0.447	0.01
	GLY	29	+0.342	0.05
	ORN	32	−0.306	0.05
	TAU	28	+0.475	0.01
Fructosamine	CIT	19	−0.585	0.01
	GLU	23	−0.543	0.01
	LEU	29	−0.362	0.05
	MET	28	−0.342	0.05
	ORN	32	−0.303	0.05
	PHE	32	−0.318	0.05
	TYR	32	−0.355	0.05
	VAL	26	−0.340	0.05
GHb	ALA	28	+0.339	0.05
	GLY	29	+0.314	0.05
	PHE	32	−0.495	0.01
	TRP	30	−0.336	0.05
	TYR	32	−0.466	0.01
BLOOD PLASMA				
Glycaemia	CAR	26	−0.364	0.05
	GLY	24	−0.415	0.05
	ILE	26	+0.386	0.05
Glycosuria	ARG	14	−0.461	0.05
	CAR	26	−0.511	0.01
	MET	25	−0.395	0.05
	PHE	27	−0.550	0.01
	PRO	14	−0.627	0.01
	TYR	27	−0.326	0.05
Fructosamine	VAL	25	−0.353	0.05
GHb	ALA	24	−0.398	0.05
	TAU	24	−0.346	0.05
	THR+SER	24	−0.347	0.05

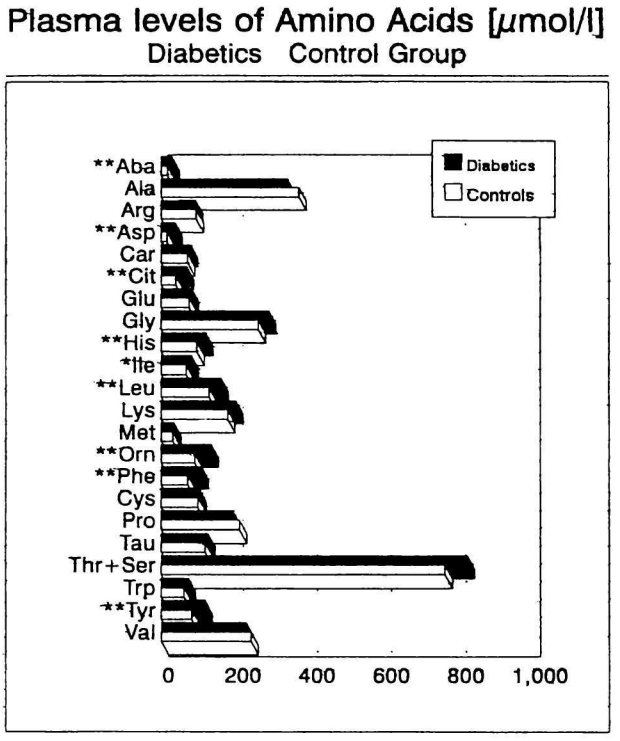

Fig. 1.

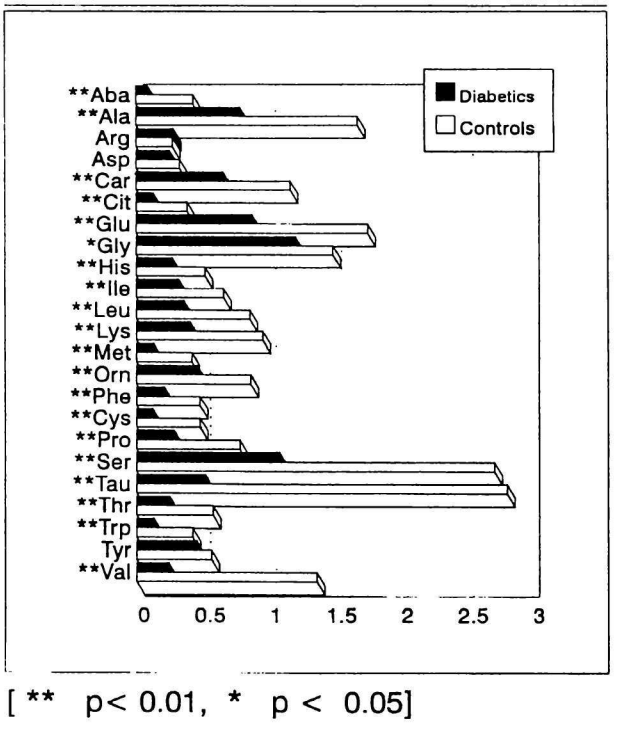

Fig. 2.

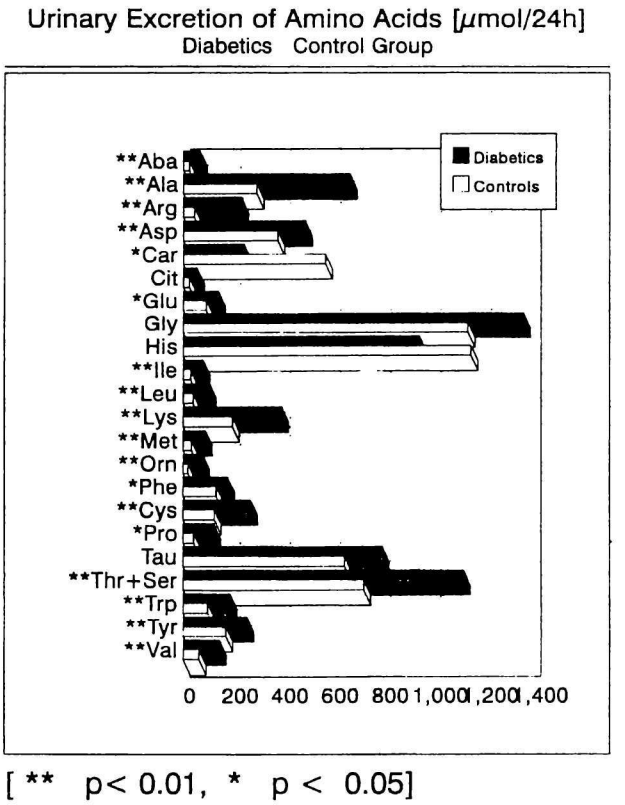

Fig. 3.

RESULTS

The concentrations of AA in plasma, erythrocytes and urine in patients with DM I and the control group are shown in Fig. 1–3. Only statistically significant correlations between glycosylated hemoglobin (GHb), glycaemia, glycosuria, fructosamine and AA levels in red cells, urine (24 h) and blood plasma are shown in Table 1.

DISCUSSION

The kidney plays a major role in the regulation of many body pools of AA through synthesis, degradation and/or urinary excretion. In the patients with DM I most of urinary AA was excreted in higher levels than in controls (Fig. 3). In spite of this and treatment by insulin [2, 3] some AA (Aba, Asp, Cit, His, Ile, Leu, Orn, Phe, and Tyr) were in plasma increased on contrary to their decreased erythrocyte levels (compare Fig. 1 and 2). Major alterations referring BCAA and aromatic AA (Ile, Leu, Val, Phe, Tyr) maight be caused by insufficient treatment by insulin, increased gluconeogenesis, decreased proteosynthesis, and disturbances in transport system for AA [1, 4–8]. However, glycosuria did not played a substantial role in elevated losses of AA in urine.

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