Association of visfatin with inflammatory and fibrotic markers in type 2 diabetic patients with nephropathy

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INTRODUCTION AND AIMS

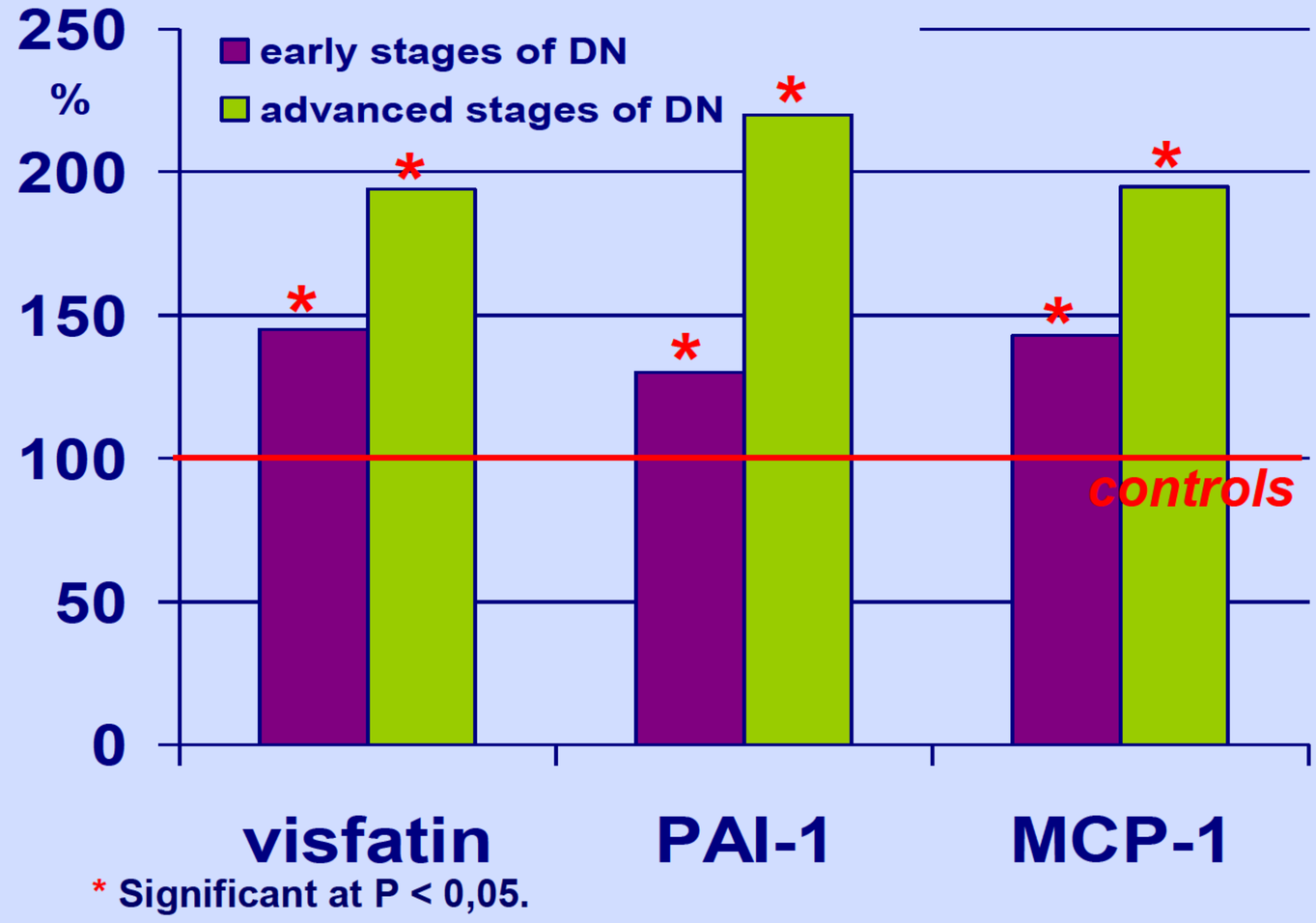
Recent studies suggest that visfatin increases the synthesis of profibrotic molecules in mesangial cells and thus may play an important role in kidney injury, however, its role in diabetic nephropathy (DN) remains to be clarified. At the same time plasminogen activator inhibitor type 1 (PAI-1) and monocyte chemoattractant protein-1 (MCP-1) are implicated in renal inflammatory and fibrotic processes, as well as in mesangial cell proliferation.

The aim of the present study was to examine visfatin correlation with markers of fibrosis and inflammation (PAI-1 and MCP-1) in type 2 diabetic patients with nephropathy.

DESIGN AND METHODS

We studied 96 type 2 diabetic patients with different stages of DN (48,8 ± 2,2 years) and 15 healthy control subjects with the same age and sex. Concentrations of plasma visfatin, PAI-1 and MCP-1 were determined by enzyme-linked immunosorbent assay. We investigated the association between plasma visfatin level with PAI-1 and MCP-1. We also tested the relationship between visfatin concentrations, urinary albumine and serum creatinine.

Fig.1. Visfatin, PAI-1 and MCP-1 percentage in blood plasma of patients with diabetic nephropathy compared with healthy controls



* Significant at P < 0,05.

Table 1. Correlation coefficients (r) of visfatin with PAI-1, MCP-1 urinary albumin, serum creatinine and glomerular filtration rate (GFR)

	visfatin, pg/ml
PAI-1, pg/ml	0.68
MCP-1, ng/ml	0.74
urinary albumin, g/l	0.63*
serum creatinine, μmol/l	0.67*
GFR	- 0.58*

* Significant at P < 0,05.

RESULTS

Our results showed that the development of DN is accompanied by significant growth of visfatin, PAI-1 and MCP-1 in plasma when compared with healthy control subjects. Visfatin, PAI-1 and MCP-1 levels were consistently associated with nephropathy progression. Their levels increased from early to advanced disease stages (visfatin, P=0.01; PAI-1, P=0.02; MCP-1, P=0.001). The mean plasma visfatin level in patients with nephropathy was (42.7±4.2) ng/ml, controls – (22.5±1.9) ng/ml. Visfatin correlated positively with PAI-1 and MCP-1 (rho = 0.68; P < 0.03 and rho = 0.74, P < 0.01 respectively).

In all patients, there were significant positive correlations between urinary levels of albumine, serum creatinine and plasma visfatin.

CONCLUSION

These findings suggest that DN progression is closely associated with visfatin elevation. Plasma visfatin is strongly associated with inflammatory and fibrotic markers MCP-1 and PAI-1 in DN patients. Thus, visfatin might be involved in the complex interactions between fibrosis, inflammation, and glomerulosclerosis and their major clinical consequences; however, further prospective studies are required to prove this hypothesis.

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