PLASMA CONCENTRATIONS OF VITRONECTIN IN PATIENTS WITH
GLomerulonephritis

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Abstract

We measured the plasma concentration of vitronectin by enzyme-linked
immunosorbent assay (ELISA) in patients with various renal diseases and
compared them with those in normal controls. In patients with IgA
nephropathy (IgA GN) (284.8 ± 77.4), and membranous nephropathy (MN)(310.4 ±
84.8) without renal insufficiency, the plasma concentrations of vitronectin
showed a significant increase compared with normal controls (231.8 ±
50.9)(p<0.05). In patients with chronic renal failure (CRF) without
hemodialysis (HD) (227.7 ± 91.3), the plasma concentrations of vitronectin
were significantly lower than in patients with IgA GN (p<0.01) and MN
(p<0.05) without CRF. In HD patients (180.9 ± 42.3), the plasma
concentrations of vitronectin were significantly lower than in normal
controls (p<0.05). These results suggested that the plasma concentration of
vitronectin may reflect the damage to renal tissue and progression in
glomerulonephritis.

Key words; vitronectin, glomerulonephritis, hemodialysis, chronic renal

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failure, enzyme-linked immunosorbent assay (ELISA)

INTRODUCTION

Vitronectin (S-protein) is a glycoprotein that has many functions, such as the regulation of the complement system, coagulation system and promotion of cell attachment (1). Previously it was reported that vitronectin was increased in the glomeruli in glomerulonephritis, and vitronectin was supposed to play an important role in glomerulonephritis (2, 3). However, there are few reports on investigation of the changes in the plasma vitronectin concentration in renal diseases (4). In this study, in order to investigate the changes in the plasma concentration of vitronectin in patients with glomerulonephritis, the plasma vitronectin concentration was measured by the ELISA system in patients with glomerulonephritis and renal insufficiency.

PATIENTS AND METHODS

Vitronectin was measured with a sandwich ELISA kit (VN test kit, Iwaki Glass Co. Ltd, Chiba, Japan) with two monoclonal antibodies (5). Twenty-seven patients with chronic glomerulonephritis without chronic renal failure (serum creatinine levels were defined as less than 1.5 mg/dl), who consist of 20 patients with IgA nephropathy (IgA-GN) and 7 patients with membranous nephropathy (MN), 43 patients with chronic renal failure (CRF) (serum creatinine levels were defined as more than 1.5 mg/dl) who include 14 patients under hemodialysis (HD), and 16 healthy volunteers admitted to the present study. Patients with additional diseases associated with malignant disorders or liver diseases or infectious diseases were excluded from the
Figure 1 Plasma concentration of vitronectin in renal diseases (mean±SD).
Significantly different at ** p<0.01, * p<0.05
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study. We used the Wilcoxon test to analyze the data. All values were expressed as mean ± SD.

RESULTS

In patients with IgA GN (284.8 ± 77.4) and MN (310.4 ± 84.8) without CRF, the plasma concentration of vitronectin was significantly higher than normal controls (231.8 ± 50.9)(p<0.05). The plasma concentration of vitronectin in patients with CRF without HD (227.7 ± 91.3) was significantly lower than in patients with IgA GN (p<0.01) and MN (p<0.05) without CRF. In patients with HD (180.9 ± 42.3), the plasma concentration of vitronectin was significantly lower than in normal controls (p<0.05)(Figure 1).

DISCUSSION

Vitronectin is a 75 kDa glycoprotein which has many functions such as regulation of the terminal step of the complement activation system, regulation of blood coagulation by binding to heparin and thrombin anti-thrombin III complex, regulation of the fibrinolytic system by binding to plasminogen activator inhibitor-1, and promotion of cell attachment (1). Vitronectin is present in serum, plasma, and various tissues such as the extracellular matrix. Vitronectin has been reported to be produced by macrophages, fibroblasts and hepatocytes (1, 6). Previously, the plasma concentration of vitronectin was reported to be increased in patients with malignant disorders and infectious diseases and decreased in severe liver diseases such as liver cirrhosis, and disseminated intravascular coagulation (7, 8, 9).

In the present study the plasma vitronectin concentration was measured
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In patients with chronic glomerulonephritis, CRF, and HD. In patients with immune complex type chronic glomerulonephritis (IgA GN and MN), the plasma vitronectin level was significantly higher than in normal controls. Vitronectin was reported to be produced by monocytes and macrophages as well as in the liver. In patients with IgA GN and MN, the production of vitronectin by these cells is increased.

Lower levels of plasma vitronectin in patients with CRF without HD may reflect the progression of renal damage in glomerulonephritis. Accumulation of vitronectin in sclerotic glomeruli and atherosclerotic and fibrotic lesion were reported (10, 11). With the progression of glomerulonephritis, the number of sclerotic glomeruli increases and interstitial lesions of fibrosis spread. A decrease in plasma vitronectin in patients with CRF may be due to its consumption in damaged glomeruli and fibrotic lesions in interstitium. Denaturization of vitronectin by uremic toxins such as urea N and guanidine is also a possible cause of a decrease in plasma vitronectin.

The lower plasma vitronectin concentration in patients under HD may be due to its consumption for complement activation during regular HD, or its denaturization by uremic toxins and binding to heparin.

The present study indicates that vitronectin plays an important role in the progression of glomerular sclerosis and interstitial fibrosis in glomerulonephritis, and the plasma vitronectin concentration may reflect the progression of glomerulonephritis.

References

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