

The Influence of Angiotensin-Converting Enzyme Inhibitor Lisinopril (LN) in Patients with Idiopathic Membranous Nephropathy (IMN) and Nephrotic Syndrome (NS)

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Introduction

Idiopathic membranous nephropathy (IMN) remains the most common cause of the nephrotic syndrome (NS) in adults world wide. That is why IMN is the second or third most common cause of end stage renal disease within the primary glomerulonephritis group. The management of IMN has to do with all the factors that lead to glomerular injury and in chronic renal failure(1). Specific therapy such as immunosuppressive drugs have been used in treatment of IMN with NS. Before considering the so-called specific therapy it is important to use a non-toxic therapy that includes the management of edema, hyperlipidemia, hypertension and hypercoagulability as well. Angiotensin converting enzyme (ACE) inhibitors are significantly useful in reducing systemic and intraglomerular hypertension. They also have effect in the remission of urine protein excretion (2,3). The role of ACE inhibitors in proteinuria and renal injury has been studied in experimental models of IMN (4). During the non-specific therapy of IMN the treatment with HMG CoA reductase inhibitors has also been useful in combination with ACE inhibitors, influencing the inflammatory and fibrogenic processes (5).

Our study has been held out to evaluate the effects of ACE inhibitor lisinopril (LN) in patients with IMN and NS.

Materials and methods

The aim of our study was to evaluate the influence of angiotensin converting-enzyme inhibitor, lisinopril (LN) in patients with IMN and NS. Thirteen patients, 6 male and 7 female, aged 32.1±15.3 years with IMN and NS were treated with 5-10 mg/day of LN for a period of nine months. Serum creatinine (CR), albumin (AL), total cholesterol (CHO), 24 hour proteinuria (PR) and mean arterial pressure (MAP) were considered in all patients at baseline and at the end of months 1, 3, 5, 7, 9 during a nine month therapy. The results are expressed mean±SD. Statistical analysis was performed with the use of ANOVA. Values less than 0.05 were considered to be significant.

Results

The results of statistical analysis by ANOVA for repeated measures are shown in the table

	CR(mg/dl)	AL(g/dL)	CHO(mg/dL)	PR(g/24h)	MAP(mmHg)
Baseline	1.27±0.48	2.27±0.41	347.38±81.44	4.82±2.26	107.15±11.93
Months					
1	1.25±0.43	2.50±0.48	342.76±108.6	2.97±1.08	95.76±7.18
3	1.26±0.40	2.63±0.50	326.23±89.04	2.39±1.05	95.15±7.43
5	1.27±0.41	2.70±0.55	313.76±90.17	2.15±0.81	94.92±6.40
7	1.27±0.39	2.93±0.52	284.23±92.08	2.03±0.93	94.61±6.91
9	1.30±0.39	3.09±0.59	276.53±69.92	1.83±0.72	95.61±6.42
p: 1-baseline	NS	0.002	NS	0.002	<0.001
p: 3-baseline	NS	<0.001	NS	0.001	<0.001
p: 5-baseline	NS	<0.001	0.013	0.001	<0.001
p: 7-baseline	NS	<0.001	<0.001	<0.001	<0.001
p: 9-baseline	NS	<0.001	<0.001	<0.001	<0.001

It should be noticed that despite the remission of PR and the increase of AL, ten of our patients had PR higher than

1.5 g/24h and the six of them had AL lower 3 g/dl at the end of the study.

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Conclusions

In patients with idiopathic membranous nephropathy and nephrotic syndrome treatment with lisinopril reduces 24h proteinuria and increases serum albumin levels from the first month of therapy. It also results in reduction of cholesterol levels since fifth month of treatment. No change in renal function was developed during therapy. These changes seem to be related with the control of the mean arterial pressure.

References

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