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LETTER TO THE EDITOR

Aliskiren as an antiproteinuric add-on therapy in primary membranous nephropathy

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To the Editor:

The management of subnephrotic proteinuria in membranous nephropathy is challenging. We retrospectively analyzed fifteen biopsy-proven membranous nephropathy adults with proteinuria of 1–3 g/day (mean 2.4 g/day) and mean serum creatinine of 0.8 mg/day. Secondary causes were ruled out. All the cases were on conservative therapy receiving angiotensin-converting enzyme inhibitors (ACEI) and angiotensin receptor blockers (ARB). The mean blood pressure was 124/82 mm/Hg and mean serum potassium was 4.0 meq/l. Aldosterone antagonists or receptor blockers were not used. After six months of ACEI and ARB, nine cases were still proteinuric in the range of 1–3 g/day. Aliskiren (150 mg/day) was added and titrated to a maximum tolerated dose of 300 mg/day. Three months later, in eight cases mean blood pressure was 120/76 mm/Hg and mean proteinuria decreased to <500 mg/day which was independent of the systemic blood pressure-lowering effect of the drug (Table 1). The cases were followed for additional three months. The benefits continued and there was no relapse of the disease or worsening of renal functions. None of the cases developed side-effects needing the drug to be stopped.

This is a preliminary analysis of the use of aliskiren in membranous nephropathy. It highlights the fact that despite adequate blood pressure control and use of ACEI and ARB, there is a need to block renin angiotensin pathway by other steps. Aliskiren interferes with first and rate-limiting step in renin enzyme cascade. The high specificity of renin for its substrate reduces likelihood of unwanted interactions and side-effects. Preliminary data in humans show that aliskiren reduces blood pressure and proteinuria [1]. It has also been found useful in hypertensive diabetics with proteinuria [2]. The antiproteinuric benefit is likely to be related to both lowering of intraglomerular pressure and the anti-inflammatory/anti-proliferative effects of the drug.

Our analysis has some limitations. The types of ACEI and ARB were based on the physician's preference and aldosterone blockade was not achieved in any of the cases. Second, we had a small sample size and short duration of follow up. A larger randomized controlled trial is needed to fully explore the benefits of aliskiren in proteinuric diseases.

To conclude, aliskiren has proved a useful adjunct to the combination of ACEI and ARB in management of membranous nephropathy and the benefits may possibly be extrapolated to other glomerular diseases.

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Table 1 Table showing results for the nine patients before and 3 months after add-on of aliskiren

Patient no.	Age, sex	Systolic BP (mmHg)	Diastolic BP (mmHg)	Proteinuria (g/day)	S. creatinine (mg/dayl)	S. potassium (meq/l)
1	24, M	126/122	88/82	2.9/0.7	0.9/1.0	4.2/4.3
2	32, M	122/122	86/76	1.9/0.4	0.6/1.0	3.8/3.9
3	36, M	120/114	74/64	1.0/0.1	1.0/1.1	4.6/4.5
4	28, M	126/124	86/80	2.8/0.6	0.7/0.7	4.1/4.0
5	36, F	126/120	80/80	2.3/0.5	1.1/1.0	3.6/3.8
6	46, M	130/124	84/80	2.9/0.2	0.6/0.7	3.7/4.1
7	23, M	122/116	76/70	2.7/0.7	0.7/1.0	4.0/4.0
8	26, M	124/118	82/76	2.1/0.2	0.6/0.7	3.8/3.7
9	54, F	120/120	82/74	3.0/2.5	1.0/1.3	4.2/4.7

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