NDT Plus (2008) 5: 376

However, despite the well-known antiproteinuric effect of Ang II antagonists [2–6], in none of these cases was a similar treatment undertaken alone. We feel that in our patient Ang II antagonists were credited with an unexpected success and avoided the toxic effects of corticosteroids and/or of immunosuppressive medications. We admit that a spontaneous remission cannot be ruled out, as it has been observed in up to one-third of adults with MCD [7,8]. However, in such a case it is slowly obtained and requires a mean time of 79 weeks [7]. This leads to believe that Ang II antagonists were the best explanation for the rapid remission in our patient. This case prompts us to suggest that Ang II antagonists should be systematically tried in MCD and that corticosteroids might be avoided with this symptomatic first line treatment, a treatment that has the advantage of being devoid of major side effects.

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The pattern of proteinuria following karate (Kumite) competitions

Sir,

Despite the large amount of proteins in the plasma, the urine is virtually protein free due to the selectivity of the glomerular barrier [1]. The major component of urinary protein is a tubular protein (Tamm-Horsfall) while albumin constitutes 30–40% of the total urinary protein. Various physiologic settings, including exercise, can induce a transient increase in the urinary protein excretion that is usually benign and reversible. The type of post-exercise proteinuria depends on the intensity of exercise rather than its duration, so that moderate exercise induces glomerular and heavy exercise glomerular-tubular mixed-type protein loss [2]. Post-exercise proteinuria may be due to the loss of the charge selectivity from the glomerular capillary wall, a relative preservation of the glomerular filtration rate, proteinuria out of proportion of maximal tubular reabsorption capacity following heavy exercise [2,3] and oxidative stress produced by free radicals due to enhanced oxygen consumption in muscles [4]. The purpose of our study was to compare the amount and pattern of proteinuria before and after karate (Kumite) competition in 18 male practitioners, aged 18–21 years, with similar physical characteristics. All practitioners competed in three rounds, each lasted 3 min, with a 10 min resting interval between them. Urine samples were collected just before the competition and during 24 h thereafter. Total urinary protein, urinary beta2-microglobulin B2M as tubular and albumin as glomerular protein were assayed. Before competition, the mean value of total urinary protein, albumin and B2M as basal levels were 70.68 ± 12.5, 4.84 ± 3.17 and 0.0217 ± 0.0133 mg/day, respectively. After competition, the mean values of 24-h total urine protein (196.05 ± 70.88 mg/day), albumin (34.07 ± 32.88 mg/day) and B2M (0.0933 ± 0.0372 mg/day) levels were significantly increased ($P = 0.023$, $P = 0.001$ and $P < 0.001$). This study revealed significantly increased proteinuria of a mixed type (albumin and B2M) in all practitioners following exhaustive short-term competition. Despite the mixed type of proteinuria, we observed a six-fold increase compared to the basal level in urine albumin (versus four-fold increase for urinary B2M) suggesting a more prominent role of glomerular proteinuria probably due to glomerular membrane permeability changing factors, such as sympathetic overactivity and competitive stress.

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The effect of angiotensin type 1 receptor blockade on adhesion molecules in patients with IgA nephropathy

Sir,

Various studies have linked inflammation and endothelial dysfunction in patients with chronic renal disease [1]. Plasma levels of adhesion molecules [soluble intracellular adhesion molecule-1 (sICAM) and soluble vascular adhesion molecule-1 (sVCAM)] are markers of endothelial dysfunction and also risk factors for cardiovascular disease in patients with IgA nephropathy (IgAN) [2]. It is known that vascular lesion begins long before its clinical manifestation and its pathogenesis involves endothelial dysfunction and low-grade inflammation. Numerous studies provide evidence that ACE inhibitors or angiotensin II receptor antagonists (ARBs) are more effective than other antihypertensive drugs in slowing the progressive decline in glomerular filtration rate in IgAN [3]. Our aim was to test whether blocking the renin–angiotensin system (RAS) with irbesartan decreases levels of adhesion molecules in patients with biopsy-proven IgAN.

We included in our study 36 patients (M/F 26/10, 51 ± 12.5 years). The inclusion criteria were biopsy-proven IgAN (defined by standard morphologic and immunohistochemical criteria), serum creatinine $\leq$1.5 mg/dl and urinary protein excretion $\geq$500 mg/day in at least three consecutive determinations during the previous 6-month period. Exclusion criteria were diabetes mellitus, coronary artery disease, peripheral vascular disease, stroke, acute infection or the inflammatory process on course and marked hypercholesterolaemia. Blood samples were collected from all patients before (*T*0) and after 16 weeks (*T*1) of treatment with 300 mg of irbesartan given once daily in the morning. A thorough blood chemistry control was performed in all patients. Creatinine clearance was determined by using venous blood for serum levels of creatinine and a 24-h urine collection. Serum and urinary creatinine concentration was measured using the Jaffé method. Serum intercellular adhesion molecule-1 and vascular cell adhesion molecule-1 were measured by immunosorbent assay (ELISA). Statistical analyses were performed using SPSS version 13.0 (SPSS, Chicago, IL, USA).

Systolic (SBP) and diastolic blood pressure (DBP) was significantly lower after irbesartan was given (from SBP *T*0: 144 ± 19 mmHg DBP *T*0: 93 ± 9 mmHg to SBP *T*1: 129 ± 9 mmHg DBP *T*1: 88 ± 8 mmHg, $P < 0.01$). Proteinuria levels were also significantly lower after treatment: from 1.6 ± 0.7 g/24 h − *T*0 to 1.1 ± 0.9 g/24 h − *T*1 ($P < 0.001$). We observed a significant decrease of sICAM and sVCAM plasma levels in patients after treatment with irbesartan (sICAM *T*0: 628 ± 163 ng/ml to sICAM *T*1: 369 ± 112 ng/ml, $P < 0.001$; sVCAM *T*0: 1028 ± 649 ng/ml to sVCAM *T*1: 688 ± 248 ng/ml, $P < 0.001$) (Table 1).

Table 1. Summary of before (T0) and (T1) the treatment in each parameter (BP, sICAM and sVCAM)

Parameter	*T*0	*T*1	*P*
SBP (mmHg)	144 ± 19	129 ± 9	$P < 0.01$
DBP (mmHg)	93 ± 9	88 ± 8	$P < 0.01$
Proteinuria (g/24 h)	1.6 ± 0.7	1.1 ± 0.9	$P < 0.001$
sICAM (ng/ml)	628 ± 163	369 ± 112	$P < 0.001$
sVCAM (ng/ml)	1028 ± 649	688 ± 248	$P < 0.001$

SBP, systolic blood pressure; DBP, diastolic blood pressure; sICAM, soluble intracellular adhesion molecule-1; sVCAM, soluble vascular adhesion molecule-1.

Studies [2,4] have proven the important role of inflammation in the outcome of IgAN. Angiotensin type 1 (AT1) receptor antagonist significantly decreases proteinuria and slows renal deterioration in patients with IgAN. Our data suggest that treatment with irbesartan in patients with IgAN has a beneficial effect on inflammatory markers. The interfering with the inflammatory markers of AT1 antagonist could, at least partially, explain the effect of AT1 receptor blockade on renal survival in patients with IgAN. It is obvious that additional studies are needed to verify this hypothesis, especially in order to rule out the direct effect of lowering blood pressure on the inflammatory markers taken into consideration.

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