NEFROLOGÍA. Vol. XXIV. Número Extraordinario (I). 2004

Terapia organoprotectora antihipertensiva para pacientes con enfermedad renal e hipertensión

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Patients with renal disease not only run a great risk of end-stage-renal failure, but also high risk for cardiovascular morbidity and mortality. Indeed, a decreased glomerular filtration rate or a excessive leak of proteins in the urine are prime risk markers for CV morbidity and mortality. As far as protein leaks are concerned, even low range leakage called microalbuminuria (30-300 mg/day) appears to be such a marker.

The classical cardiovascular risk profiling is based on glucose metabolism (diabetes), blood pressure, cholesterol, smoking (overweight, and lifestyle). These parameters not only allow us to classify the risk of a patient in the office, but they also allowed us to justify our therapeutic strategies. These therapies are aimed at reducing these risk factors/markers. Indeed, metabolic control of glucose was a big step in preventing the life threatening end-organ effects of type 2 diabetes. Even more impressive effects were seen when one controls blood pressure on end-organ damage. Recently, lowering of cholesterol has been added to this cardiovascular protective armamentarium.

By far the biggest additive step has been made recently by the introduction of tools to intervene in the renin-angiotensin-aldosteron system, such as ACE-inhibitors and AII-antagonists. These drugs add to cardiovascular protection beyond blood pressure and blood glucose control (HOPE, LIFE, CHARM).

Interestingly, renal function as well as urinary protein excretion independently contribute to the above classical risk factors for CV morbidity and mortality. In fact, serum creatinine (GFR) and proteinuria are in many studies both in the general population, as well as in other risk groups such as hypertensives, the strongest predictors of CV risk.

Recent data have shown that if one targets the kidney with renoprotective antihypertensive therapies such as ACE-inhibitors and AIIA antagonist (IRMA2, IDNT, RENAAL), one may also protect the heart. In fact, unpublished data show that the more one protects the kidney the more one protects the heart. Thus, not only are patients with a compromised renal function (either low GFR or high proteinuria or both) at markedly higher CV risk, they also are responding as good or even better to CV protective therapy.

These data urge us to address the question: should we not target and titrate our therapy to improving the kidney to obtain a maximal effect on CV risk. This question is not only relevant in secondary prevention, but certainly also in primary prevention. Data will be presented to make that case.

This opens the door for multi-organ disease management and therapy in an ever growing disease