Further insights into the renal effects of drugs that disrupt the renin–angiotensin system

Results of two large, retrospective, cohort studies further our understanding of the long-term effects of angiotensin-converting-enzyme (ACE) inhibitors and angiotensin II type 1 receptor blockers (ARBs) in renal transplant recipients and diabetic patients.

Of the 1,250 renal transplant recipients in the Austrian Dialysis and Transplant Registry who received ACE inhibitors or ARBs, 74% survived to 10 years after transplantation and 59% had surviving grafts at this time, compared with only 53% and 41% of those who did not receive these drugs ($n=781$). Treatment with ACE inhibitors or ARBs might, therefore, not only aid graft recovery immediately after kidney transplantation, but also improve long-term recipient and graft survival.

Results of a Canadian 16-year follow-up study of progression to renal disease in patients with diabetes ($n=6,012$) show ACE inhibitors in a less favorable light. After 3 years, the incidence of end-stage renal failure was greater in patients receiving ACE inhibitors than in those receiving thiazide diuretics (adjusted rate ratio of renal failure 4.2). By contrast, neither β -blockers nor calcium antagonists increased the rate of renal failure relative to that seen with thiazide diuretics. Controversially, the researchers propose that, in the long-term, ACE inhibitors might be contributing to the increased incidence of end-stage renal disease worldwide.

It is doubtful that clinical practice will change directly as a result of either of these two studies; the findings must first be confirmed in randomized, controlled trials.

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Original articles Heinze G *et al.* (2006) Angiotensin-converting enzyme inhibitor or angiotensin II type 1 receptor antagonist therapy is associated with prolonged patient and graft survival after renal transplantation. *J Am Soc Nephrol* 17: 889–899
 Suissa S *et al.* (2006) ACE-inhibitor use and the long-term risk of renal failure in diabetes. *Kidney Int* 69: 913–919

Pediatric patients on hemodialysis experience increased arterial stiffness

Abnormalities in arterial wall structure are associated with end-stage renal disease in children, as well as in adults, according to a recent study.

This is reportedly the first time that such an association has been shown in children, and helps to explain the high incidence of cardiovascular complications in children with chronic kidney disease.

Comparison of 14 children on hemodialysis (mean age 14.1 ± 2.6 years) and 15 matched controls showed that uremic patients had increased arterial stiffness (measured as aortic pulse wave velocity), peripheral arterial reflectivity (measured as aortic augmentation index) and atheromatous disease (measured as carotid intima–media thickness)—parameters associated with morbidity and mortality. Children requiring hemodialysis had thicker left ventricular walls, and a greater left ventricular mass index.

The results indicate that uremia has a notable effect on the structure, and thereby function, of arteries even at a young age, increasing the risk of atherosclerosis/arteriosclerosis. Comparison of measurements taken before and after a single hemodialysis session showed that dialysis did not have a positive effect on either aortic augmentation index or pulse wave velocity, indicating that only structural alterations, and not functional alterations, lead to increased arterial stiffness.

An annual reassessment of these patients is planned, both while they are on dialysis and after they have undergone transplantation, to correlate changes in baseline parameters with survival and left ventricular alterations.

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Original article Covic A *et al.* (2006) Increased arterial stiffness in children on haemodialysis. *Nephrol Dial Transplant* 21: 729–735

Determining the cause of fast transport status in peritoneal dialysis patients

Fast transport status (FTS) is associated with worse outcome in dialysis patients. It is important to determine whether FTS in a particular patient is due to inflammation or other causes—therapeutic strategies such as icodextrin administration and automatic peritoneal dialysis can improve the outcome if FTS is caused by an anatomic large surface area, but if FTS is due to inflammation the underlying cause of the inflammation must be treated. A recent study has compared the abilities of the