Cystatin C is 'better than' creatinine for GFR estimation in adults with cystic fibrosis

Malnutrition is common among patients with cystic fibrosis (CF), which could affect creatinine-based calculation of glomerular filtration rate (GFR). Beringer and colleagues have investigated whether cystatin-C-based estimation is a better alternative.

GFR was measured by use of iothalamate clearance and estimated on the basis of serum creatinine level (Modification of Diet in Renal Disease [MDRD] equation) and serum cystatin C level (100/cystatin C – 14 ml/min/1.73 m^2) in 19 patients with CF (aged 24–48 years). In addition, creatinine clearance was calculated by use of the Cockcroft–Gault formula.

Serum creatinine levels were lower in patients with CF than in 19 age-matched healthy controls, but serum cystatin C level and iothalamate-measured GFR were both similar in the two groups. Cystatin C-estimated GFR had the narrowest 95% limits of agreement and was accurate to within 30% of measured GFR on the greatest percentage of occasions (78.9%). No significant differences were noted in 50% accuracy level or bias between the three prediction methods.

At a cut-off of 90 ml/min/1.73 m^2, the area under a receiver operating characteristic curve was greater for cystatin-C-estimated GFR (0.93) than for Cockcroft–Gault-estimated creatinine clearance (0.56; $P = 0.005$) or MDRD-estimated GFR (0.54; $P = 0.003$). The area under the curve was also slightly greater for cystatin-C-estimated GFR at a cut-off of 80 ml/min/1.73 m^2.

The use of cystatin C to estimate GFR should be considered in patients with CF, particularly in those who are at risk of renal disease.

Original article Beringer PM *et al.* (2008) GFR estimates using cystatin C are superior to serum creatinine in adult patients with cystic fibrosis. *J Cyst Fibros* [doi:10.1016/j.jcf.2008.07.004]

Inability to monitor renal function is not a contraindication to first-line HAART

Highly active antiretroviral therapy (HAART) seems to improve renal function in patients with HIV; however, some antiretroviral drugs require dose adjustment in the setting of renal dysfunction and others can even cause renal impairment. Peters *et al.* have evaluated the importance of monitoring renal function in a rural population of patients on HAART.

The cohort comprised 508 HIV-positive Ugandan adults with symptomatic disease or a CD4 cell count not exceeding 250 cells/mm^3 (median age 39 years; 59% female) who had been monitored for at least 2 years after initiation of HAART with stavudine plus lamivudine and either nevirapine or efavirenz, and had undergone measurement of serum creatinine level at 1 year and 2 years. Individuals with a baseline Cockcroft–Gault-estimated creatinine clearance <25 ml/min were excluded.

At baseline, 8% of participants had an elevated serum creatinine level (≥133 μmol/l) and 20% had reduced renal function (creatinine clearance 25–50 ml/min). During the follow-up period, the median serum creatinine level declined by 16% and the median creatinine clearance increased by 21% ($P < 0.0001$ for both). Among patients with reduced renal function, the improvement was more marked; median creatinine clearance increased by 53% ($P < 0.0001$). *De novo* reduction of creatinine clearance (to ≤50 ml/min) occurred in 19 patients during the study.

The authors conclude that in settings where regular monitoring of renal function cannot always be performed, first-line HAART can initially be dosed simply on the basis of body weight.

Original article Peters PJ *et al.* (2008) Antiretroviral therapy improves renal function among HIV-infected Ugandans. *Kidney Int* **74:** 925–929

MYH9 is associated with kidney disease in African Americans

MYH9 encodes a chain of non-muscle myosin and is expressed in podocytes. Two studies published recently in *Nature Genetics* have reported that genetic variation at the *MYH9* locus of chromosome 22 can explain much of the increased risks of end-stage renal disease and focal segmental glomerulosclerosis (FSGS) among African Americans.

Kopp *et al.* performed genome-wide mapping by admixture linkage-disequilibrium (MALD) in 190 African Americans with FSGS and 222 African American controls without