Measuring blood levels of the protein cystatin C can help identify individuals with chronic kidney disease (CKD) who have a poor prognosis, new research finds (Peralta CA, et al. Cystatin C identifies CKD patients at higher risk for complications. J Am Soc Nephrol, January 2011).

“The manuscript nicely describes potential methods for incorporation of cystatin C into clinical practice, specifically as a confirmatory test for CKD,” said Lesley Stevens, MD, a CKD expert at Tufts University School of Medicine who was not involved with the research.

The findings could impact the care of many patients, because CKD affects millions of adults in the United States, and its prevalence is rising, particularly in the elderly.

Beyond creatinine

To assess kidney function, doctors most often measure an individual’s blood levels of creatinine, a breakdown product that is produced by muscles and is filtered by the kidneys. Creatinine tests are imperfect, however, because creatinine levels can vary with muscle mass and protein intake. In addition, creatinine tests cannot accurately detect mild kidney impairment.

“We need a more accurate approach to identifying persons with reduced kidney function. Relying on a serum creatinine test alone leads healthy individuals to erroneously be identified as having chronic kidney disease,” said Andrew Rule, MD, of the department of nephrology and hypertension at the Mayo Clinic.

Serum tests of cystatin C, a protein that is filtered from the blood by the kidneys, have emerged as an alternative test of kidney function that

CKD Children Born Small Have Trouble Catching Up

A new study affirms what some pediatric nephrologists already suspected: Children with chronic kidney disease (CKD) who have low birth weight or are born small for gestational age (SGA) may not have the normal “catch-up” growth seen in other children who are born small.

Of course, children with kidney disease are well-known to be at high risk for growth problems.

“But if they’re low birth weight or SGA, they’re even more likely to have poor growth than other kids with CKD, even correcting for severity of kidney disease, number of years they’ve had kidney disease, and the type of kidney disease,” said Larry Greenbaum, MD, PhD, lead author of the new report. The paper appears in the January 2011 issue of Clinical Journal of the American Society of Nephrology.

Based on prospective follow-up in a large sample of children with CKD, the data also show higher than usual rates of low birth weight and SGA birth even in children who do not develop kidney disease until long after birth.

“That suggests it’s possible that being born low birth weight or SGA may increase the likelihood of developing an acquired kidney disease during childhood,” said Greenbaum.

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is less influenced by muscle mass (see sidebar), p. 4. High cystatin C levels in the blood are indicative of poor kidney function, but cystatin C levels are rarely measured in the clinic.

Cystatin C in CKD

Carmen Peralta, MD, (San Francisco Veterans Affairs Medical Center and University of California, San Francisco) and colleagues studied the potential of measuring cystatin C levels to assess kidney function. Their study included 11,909 participants from the Multi-Ethnic Study of Atherosclerosis (MESA) and the Cardiovascular Health Study, two studies that were designed to investigate various aspects of cardiovascular disease and used standardized measures of kidney function.

“We hoped to illustrate the utility of combining the two filtration markers—creatinine and cystatin C—to refine the diagnosis of CKD,” said Peralta. The investigators also looked for any links between cystatin C levels and increased risks for premature death, cardiovascular events, heart failure, and kidney failure—all of which have been linked to CKD.

In MESA, 9 percent of individuals had CKD by a creatinine-based equation only, 2 percent had CKD by a cystatin C-based equation only, and 4 percent had CKD by both equations. In CHS, these percentages were 12 percent, 4 percent, and 13 percent, respectively. Compared with individuals without CKD, individuals in MESA with CKD based on creatinine had only a reduced risk of premature death, whereas individuals with CKD based on cystatin C had more than a threefold increased risk, and individuals with CKD based on both equations had nearly a twofold increased risk.

In the Cardiovascular Health Study, individuals with CKD based on creatinine only had a similar risk of premature death compared with individuals without CKD, whereas individuals with CKD based on cystatin C only had a 1.78-fold increased risk. Individuals with CKD based on both had a 1.74-fold increased risk. The pattern was similar for cardiovascular disease, heart failure, and kidney failure.

These results suggest that among adults diagnosed with CKD using the creatinine-based equation, poor prognosis is limited to patients who also have CKD based on the cystatin C equation. Therefore, cystatin C may have a role in identifying CKD patients who have the highest risk for developing complications.

“Based on our findings, we believe that cystatin C should be a confirmatory test among persons identified as having a creatinine-based estimated glomerular filtration rate below 60 mL/min/1.73 m²,” said Peralta. “Depending on the patient’s age, roughly one-third to one-half of these patients will be reassured that they do not in fact have a high risk for CKD complications.” Peralta added that although the cystatin C test is infrequently used, it has been approved by the U.S. Food and Drug Administration and is an automated blood test that is potentially available in any hospital laboratory.

“I agree with the approach suggested by Dr. Peralta and colleagues,” said Rule. “Both cystatin C and creatinine are influenced by factors other than kidney function. But by using both tests to detect a reduction in kidney function, physicians can better identify persons with chronic kidney disease who are at increased risk for death, heart disease, or the future need for dialysis or a kidney transplant.”

Study co-authors include Ronit Katz, DPhil, Ian De Boer, MD, David Siscovick, MD (University of Washington); Mark Sarnak, MD, Andrew Levy, MD (Tufts-New England Medical Center); Joachim Ix, MD (University of California San Diego); Linda Fried, MD (Pittsburgh Veteran’s Affairs Medical Center); Walter Palmas, MD (Columbia University); and Michael Shlipak, MD (University of California, San Francisco).

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