CKD: Latest on Screening, Management

Q suPAR, a new screening tool for chronic kidney disease, has gotten a lot of press recently. My practice is interested in implementing it, but we can't find information on how to obtain it. Is it commercially available yet? How can we order it? And importantly for our patients, do insurance plans cover it?

Research has been ongoing regarding biomarkers that could identify those at risk for chronic kidney disease (CKD) long before loss of renal function is apparent. A recently published study suggests that the circulating protein, soluble urokinase-type plasminogen activator receptor (suPAR), may be such a biomarker.

In a study of 3,683 subjects (ages 20 to 90) undergoing cardiac catheterization, and a further evaluation of 347 subjects in the Women's Interagency HIV Study, Hayek et al found that elevated levels of suPAR were independently associated with CKD and with accelerated loss of renal function. At five-year follow-up, 24% of the 1,335 subjects with an initial estimated glomerular filtration rate (eGFR) ≥ 60 mL/min/1.73 m^2 had developed CKD. Risk for progression to CKD was about 41% in those with a baseline suPAR level ≥ 3,040 ng/mL, compared to 12% in those with lower baseline suPAR levels.[1] Thus, the cutoff for high versus low risk appears to be 3,040 ng/mL.

Hayek and associates are not the first or the only investigators studying the connection between suPAR and kidney disease. Evolving research has suggested suPAR may be an initiating factor in the development of focal segmental glomerulosclerosis (FSGS).[2] However, a recent study did not support this association.[3]

Currently, in the United States, laboratory testing for suPAR is available only for research purposes and has not been approved by the FDA for direct patient care.[4] While more research is needed with different cohorts, there is much excitement in the field of nephrology regarding the potential role of suPAR as a biomarker for predicting CKD. **—CS**

Q I have a patient with stage 3a chronic kidney disease (glomerular filtration rate, 45-60 mL/min/1.73 m^2). I have her on a statin and an ACE inhibitor. Is there anything else I can do to slow the progression of kidney disease?

For patients with stage 3a chronic kidney disease (CKD), ongoing evaluation of risk factors and management can impact the rate of disease progression. The cornerstones of CKD care include identification and treatment of the cause; management of hypertension, albuminuria, and diabetes (if applicable); reduction of cardiovascular (CV) risk; and correction of metabolic abnormalities.[5]

When considering factors that can contribute to kidney injury, clinicians should consider possible pre-, intra-, and post-renal processes that could potentially cause injury.

Prerenal: Approximately 20% of cardiac output is directed to the kidneys. Reduced left ventricular function, diastolic dysfunction, and pulmonary hypertension can all contribute to a reduction in renal blood flow and subsequent kidney injury.[6]

Intrarenal: Exploration of possible intra-renal processes begins with a thorough history of any familial disease, hematuria, stones, proteinuria, and exposure to nephrotoxins. The nephrotoxicity profile of all medications should be examined, and patients should be educated about products, particularly OTC medications (eg, NSAIDs, common cold preparations, and herbal or weight-loss products), that can be harmful to the kidneys. Patients should also be made aware of the risk for contrast-induced renal injury, espe-

Renal Consult is edited by **Jane S. Davis, CRNP, DNP,** a member of the Clinician Reviews editorial board, who is a nurse practitioner in the Division of Nephrology at the University of Alabama at Birmingham and is the communications chairperson for the National Kidney Foundation's Council of Advanced Practitioners (NKF-CAP); and **Kim Zuber, PA-C, MSPS, DFAAPA,** a retired PA who works with the American Academy of Nephrology PAs and is also past chair of the NKF-CAP. This month's responses were authored by **Cindy Smith, DNP, APRN, CNN-NP, FNP-BC,** who practices with Renal Consultants, PLLC, in South Charleston, West Virginia, and **Crystal Johnson, PA-C, Angela Harker-Bacchus, FNP-BC, Irina Sadovskaya, PA-C,** and **Beverly Benmoussa, FNP-BC,** who practice in the Transplant Nephrology Extra-Renal CKD Clinic at the University of Michigan.

cially when considering imaging or cardiac testing. Since diabetes is a leading cause of kidney disease, good diabetic control can reduce nephropathy and slow disease progression.

Postrenal: Benign prostatic hypertrophy, kidney stones, and neurogenic bladder can all cause injury. These warrant further evaluation and treatment.

CKD often worsens existing hypertension, which is an independent risk factor for kidney failure.[7] Goal blood pressure (BP) for all patients without significant albuminuria should be < 140/90 mm Hg; for those with urinary albumin ≥ 30 mg/24 h, the goal is < 130/80 mm Hg.[8] Choice of antihypertensive agents can be tailored to other comorbidities, but an ACE inhibitor or angiotensin receptor blocker should be considered firstline treatment. Nocturnal hypertension is common in patients with CKD and an independent marker of CV risk. By dosing antihypertensive medications at bedtime, the clinician supports CV risk reduction.[9]

CKD is an independent risk factor for CV disease, thus risk factor modification should be aggressively pursued. Regardless of the cause of CKD, cigarette smoking has been associated with a more rapid decline in renal function. Patients should be counseled on the risks and offered interventions to assist in smoking cessation.[10] There is also emerging evidence that exercise likely benefits the vascular health of the kidneys and appears to slow the rate of kidney decline.[11,12] Overall, lifestyle interventions that help mitigate CV risk may directly benefit preservation of kidney function as well.

Metabolic abnormalities increase with CKD progression. Maintaining proper bone health through control of phosphate/acidosis and calcium equilibrium reduces morbidity as it relates to vascular and soft-tissue calcification. This can often be effectively managed through dietary modifications in early to moderate CKD. As the number of functioning nephrons decrease in CKD, so does the ability of the kidney to maintain proper acid/base balance. Persistent metabolic acidosis is related to CKD progression. Acid buffering with oral bicarbonate may be needed to achieve a goal CO_2 of 22 to 32 mEq/L.[8]

Through adoption of a comprehensive approach—one that is inclusive of the patient—optimal outcomes can be achieved for this rapidly growing and often underrecognized population. **—CJ, AH-B, IS, BB** **CR**

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