Findings from a new study could lead to better diagnosis and treatment of patients with immunoglobulin A (IgA) nephropathy, one of the most common diseases of the kidney. The results, published in the Journal of the American Society of Nephrology, indicate that increasing blood levels of certain autoantigens and autoantibodies may act as warning signs that a patient’s disease is worsening and that aggressive interventions are needed.

The findings also support a predominant role of an autoimmune mechanism in the pathogenesis of IgA nephropathy, which remains partly unsolved. “The intimate details of the cascade of events leading eventually to destruction of the kidneys are complex and still puzzling,” said first author Francois Berthoux, MD, of the University Hospital of Saint-Etienne, in France.

Assessing disease severity
Patients with IgA nephropathy, a condition that was first described in 1968, have increased serum levels of IgA1 that is galactose deficient. In the absence of galactose, terminal N-acetylgalactosamine residues are exposed. Consequently, such IgA1 molecules are presented as autoantigens, and IgG or IgA glycan-specific autoantibodies recognize them to form immune complexes that circulate in the blood and can settle in the kidneys. These events can damage the kidneys, which subsequently leak blood and protein in the urine. IgA nephropathy can lead to high blood pressure, swelling, and, in some cases, kidney failure.

At the time of diagnosis, it remains difficult to predict the long-term clinical outcome for patients with IgA nephropathy. “The disease is clinically heterogeneous, with 20 percent to 30 percent of patients progressing to chronic kidney disease,” said Ian Roberts, MD, of the department of cellular pathology at John Radcliffe Hospital, in England, “The challenge is to identify those patients who will progress and could potentially benefit from immunosuppressive ther-

Physician Quality Reporting System: Incentive Today, Gone Tomorrow
By Rachel Shaffer
Since 2007, physicians and other eligible health professionals have been eligible to receive bonus Medicare payments for voluntarily reporting data to the Physician Quality Reporting System (PQRS) program. Starting in 2013, that program will no longer be voluntary, and every physician and other health professional with a National Provider Identifier (NPI) number should be aware of important changes to the PQRS that will affect their Medicare payments (Table 1).

The PQRS is a congressionally mandated program operated by the Centers for Medicare & Medicaid Services (CMS). The Tax Relief and Health Care Act of 2006 first authorized the incentive program, and the Medicare Improvement for Patients and Providers Act of 2008 made it permanent. The PQRS is not entirely unique. CMS maintains several quality measurement program initiatives to help it monitor the quality of care in different environments—including the End-Stage Renal Disease Quality Incentive Program for dialysis facilities—and holds that such quality initiatives aim to give providers and patients information that improves
IgA Nephropathy

Continued from page 1

The report offers a new risk factor that, if confirmed in additional studies, can serve as a marker for selecting patients to be aggressively treated.

“The international community of pathologists and nephrologists who worked on the Oxford classification of IgA nephropathy is highly interested in finding serologic markers that could be added on the pathology score. These efforts will hopefully provide a clue for selecting IgA nephropathy patients to be treated or not and to modulate the intensity of treatment in the likely progressors,” said Rosanna Coppo, MD, who was not involved with the study and is the director of the nephropathy, dialysis and transplantation unit at Regina Margherita Children’s University Hospital in Turin, Italy. Her own work indicates that the nephrotoxicity of aberrantly glycosylated IgA1 in IgA nephropathy is enhanced in the presence of systemic signs of oxidative stress.

John Radcliffe Hospital’s Roberts, who also did not participate in the study, noted that the findings raise some important questions.

“It is unclear how the autoantibody levels change over time, and it remains to be ascertained whether levels correlate with clinical markers of activity in a longitudinal study. Another important area of future investigation is the link between autoantibody levels and histological activity in IgA nephropathy,” he said.

Study coauthors include Lise Thibaudin, MD, Nicolas Maillard, MD, PhD, Christine Mariat, MD, PhD (University Hospital of Saint-Etienne, France); Hiroyuki Yanagawa MD, PhD; Yasuhide Tomino, MD, PhD (Juntendo University, Tokyo, Japan); and Bruce Julian, MD, PhD (University of Alabama at Birmingham).

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The article, entitled “Serum autoantibodies specific for galactose-deficient IgA1 associate with disease progression in IgA nephropathy,” is available online at http://jasn.asnjournals.org/doi:10.1681/ASN.2012010053.

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