Transcript Details

This is a transcript of an educational program. Details about the program and additional media formats for the program are accessible by visiting: https://reachmd.com/programs/medical-industry-feature/igan-insights-the-impact-of-proteinuria/29182/

ReachMD
www.reachmd.com
info@reachmd.com
(866) 423-7849

IgAN Insights: The Impact of Proteinuria

Announcer:
Welcome to ReachMD. This medical industry feature, titled “IgAN Insights: Proteinuria in IgA Nephropathy” is sponsored by Novartis Pharmaceuticals Corporation. Here’s your Guest, Dr Laura Mariani.

Dr Mariani:
Hello, my name is Dr Laura Mariani, and I am a nephrologist and clinical researcher at the University of Michigan.In this video, we will discuss the impact of proteinuria on kidney outcomes in patients with IgA nephropathy.

IgA nephropathy is an autoimmune glomerulonephritis with a heterogeneous clinical presentation and variable disease progression. [1-3]

While the classically taught presentation involves visible hematuria following an upper respiratory tract or gastrointestinal infection, many patients will go on to experience a chronic progressive course marked by persistent proteinuria, with some also exhibiting glomerular inflammation.[2,4-8]

While up to 50% of patients with IgA nephropathy may already have stage 3 or greater chronic kidney disease, almost all patients are at risk of progressing within their expected lifetime.[9,10]

The multi-hit mechanism of disease encompasses several key steps in the disease pathogenesis. [9] The first 3 steps lead up to formation of galactose-deficient-IgA1–containing immune complexes, while the deposition of these immune complexes in the kidney in the fourth step involves renal injury processes.[1,9,11]

Pro-inflammatory and pro-fibrotic responses may be amplified by overactivation of the complement, endothelin, and renin-angiotensin-aldosterone systems.[1,9,11,13]

These pathological processes may then cause progressive glomerular and tubulointerstitial damage and fibrosis that manifest as proteinuria, hematuria, and chronic kidney disease and may lead to disease progression.[1,9,11,13,14]

Proteinuria is a consequence of glomerular damage, but it can also be a cause of tubular damage and progression of kidney disease through several mechanisms.[9,10,15-17]

Increases in proteinuria may be driven by:[14,19]

- Disease-specific immune-mediated processes
- Compensatory responses of healthy nephrons
- Irreversible glomerular scarring
- And loss of the protein resorptive capacity due to tubular damage

The KDIGO 2021 clinical practice guideline recommends measurement of proteinuria in patients with IgA nephropathy, as proteinuria can inform on disease progression and management.[20-22] Initiation of supportive care is recommended in patients with proteinuria to reduce the risk of progression to chronic kidney disease.[22]

A cross-sectional study was recently conducted in 606 patients with primary IgA nephropathy from the Kaiser Permanente Southern California health system. Of the patients included in the study, the mean age was 46 years, and most patients were male, and Hispanic or Latino. The study observed that the annual incidence of IgA nephropathy in the US is 1.4 cases per 100,000 person-years. Further, patients appear to be diagnosed with IgA nephropathy at advanced stages of kidney disease within our current clinical environment.[23]

Another cross-sectional study was recently conducted in the UK National Registry of Rare Kidney Diseases (or RaDaR). Analyses of eGFR slopes and kidney survival were conducted, and the authors observed that 30% of patients with a time-averaged proteinuria range of 0.44 to 0.88 g/g reached kidney failure within 10 years.[24]

Data from the retrospective study of 2299 adults and 140 children with IgA nephropathy within the UK National Registry of Rare Kidney Diseases (RaDaR). Patients enrolled had a biopsy-proven diagnosis of IgA nephropathy plus proteinuria >0.5 g/day or eGFR <60 ml/min/1.732m^2 at any time in their history of disease. Analyses of annualized eGFR slopes were calculated using linear progression to fit a straight line through patients' mean eGFR values for each of the 3-month period of follow-up. Analyses of kidney survival were conducted using Kaplan-Meier and Cox regression. Recruitment was initiated into RaDaR in 2013. Availability of patient medication and blood pressure data was a limiting factor in this study.

Announcer:
This program was sponsored by Novartis Pharmaceuticals Corporation. If you missed any part of this discussion or to find others in this series, visit ReachMD.com, where you can Be Part of the Knowledge.

FA-11324184 1/25