

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/frontlines-iga-nephropathy/the-diagnostic-journey-of-iga-nephropathy-identifying-key-obstacles/26947/>

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The Diagnostic Journey of IgA Nephropathy: Identifying Key Obstacles

Announcer:

Welcome to *On the Frontlines of IgA Nephropathy* on ReachMD. On this episode, we'll discuss the challenges in diagnosing IgA nephropathy with Dr. Gates Colbert, who's a nephrologist at the Kidney and Hypertension Associates of Dallas and an Associate Clinical Professor at Texas A&M College of Medicine. Here's Dr. Colbert now.

Dr. Colbert:

So diagnosing IgA nephropathy is a tough conundrum for most clinicians in the early stages, and this is because it's a patient that usually presents feeling perfectly well; they may occasionally have some pink urine, but their urine may have never changed any color, and they're probably being told by a primary care physician or some other clinical situation when they gave a urine study that they had some protein in their urine or some blood in their urine and the patient usually is very unaware. Additionally, they may have perfect kidney function or their GFR may have started to drop some. And so it's difficult because most patients feel very well, but their labs show up as atypical. Usually, these patients are not going on to a rapidly progressive GN or a massive drop in their kidney function or a large amount of protein and nephrotic-range proteinuria. It's usually a very mild, indolent-type presenting disease, and so this makes it difficult because the patients usually don't have any symptoms and their urine studies and their blood studies are either just mildly atypical or they may show a lot of abnormalities as well as intermittent changes. A lot of patients with IgA may go through periods where they have a good amount of protein and blood in their urine that may be associated with an illness, but then it returns back to normal, so it can be an intermittent or episodic availability to capture these studies.

So common diseases that we see in a nephrology clinic or a hypertension clinic are going to be patients who have hypertension, diabetes, or lupus, and these presentations of these disease states also can mimic IgA nephropathy presentations. Typically, you're going to see patients that maybe have preserved GFR or mildly reduced GFR, and they may have a small amount of protein or active sediment in their urine and a mild amount of blood in their urine. So these presentations, when you just look at the initial screening of bloodwork and urine studies, they're not able to determine what is the primary cause when looking at common problems like hypertension and diabetes or if it's a glomerulonephritis like lupus or Ig nephropathy. Additionally, many Americans have hypertension and diabetes, so you can have two problems at the same time. You can have hypertension and IgA nephropathy, diabetes and IgA nephropathy, or all three: diabetes, hypertension and a new diagnosis of IgA nephropathy. The way that we screen these patients can make it very difficult to discern them without doing a serologic workup and then potentially a kidney biopsy. Another problem we have is currently on the clinical market, we have almost zero biomarkers for IgA nephropathy. There is not a test that we can do in the widespread clinical setting to look for galactose-deficient IgA or some other biomarker that will signal us that a patient has IgA nephropathy.

So reminder that IgA nephropathy is going to usually start out with subnephrotic range proteinuria, maybe less than 1 gram or even 500 mg of urine protein per day and a mild amount of blood or no blood in the urine. And so this is a very common presentation that you might see for a patient that's coming in with hypertensive nephropathy diabetes mellitus, both type 1 and type 2 diabetic glomerular disease. It could potentially be another mild form of lupus nephritis or even minimal change disease, other GNs that usually start out very indolent like minimal change disease, potentially membranous or FSGS. And all these disease states can present very similarly when looking at urine studies. Additionally, a lot of those conditions start out with very preserved GFR. These are not RPGN diseases, so you're not seeing a rising creatinine over days or weeks that's very fast. It's usually going to be a very slow fibrotic and inflammatory situation. So you may see very little change from a three- and six-month visit over their lab work, and so that also makes it difficult to differentiate "Well, is this patient having diabetic nephropathy or diabetic involvement of their kidney disease, or is this an indolent IgA

that's just slowly progressive?"

Announcer:

That was Dr. Gates Colbert talking about the challenges in diagnosing IgA nephropathy. To access this and other episodes in our series, visit *On the Frontlines of IgA Nephropathy* on ReachMD.com, where you can Be Part of the Knowledge. Thanks for listening!