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To V(CUG) or Not to V(CUG) in Infants with Prenatal Hydronephrosis?

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This is a critical question that may throw even the noblest of pediatric urologists into a quandary. Our desire to help our patients simply will not allow us to stand idle and watch them “suffer the slings and arrows of outrageous fortune” that vesicoureteral reflux (VUR) can yield.¹ “To take arms against a sea of troubles” is our alternative.¹ The core of this modern-day conflict is whether diagnosing VUR in asymptomatic patients has long-term health benefits. Currently it is not clear which patients diagnosed prenatally will go on to have clinically significant VUR.^{2,3} However, we do know that not all patients with VUR are created equal. In fact, some patients have no sequelae from VUR while others have recurrent urinary tract infections (UTIs) and a minority of these patients has renal scarring.

Before the widespread use of prenatal ultrasonography, VUR was discovered in patients when they manifested clinically with febrile UTI. Radiologic investigation with voiding cystourethrography (VCUG) was required to determine the etiology of the problem and guide therapy. Prenatal ultrasonography reversed this paradigm through the identification of urinary tract abnormalities in the fetus, specifically with the detection of prenatal hydronephrosis.

Studies have linked prenatal hydronephrosis to postnatal pathology. With increasing grades of prenatal hydronephrosis, the likelihood of postnatal urinary tract pathology increases, but neither the presence nor degree of hydronephrosis on prenatal studies necessarily equates to the presence of VUR.⁴ Moreover a normal prenatal study defined by a lack of hydronephrosis does not exclude VUR.⁵ Nevertheless, prenatal hydronephrosis often triggers postnatal evaluation for VUR with VCUG. This has contributed to the 2 separate issues of over-testing and over diagnosis. Because prenatal hydronephrosis is not a good predictor of VUR, a large proportion of voiding cystourethrograms performed are negative.^{6,7} This is not inconsequential. VCUG can cause stress for the patient and parent, may incite UTI and exposes the child to radiation.^{8–10}

The idea of being able to more accurately use ultrasound findings to predict which patients need VCUG is a practical solution to over-testing. In this issue of *The Journal* Lee et al (page 914) sought to determine if any 1 of 3 additional renal and bladder ultrasonography criteria including hydroureter, renal dysmorphism and/or duplication could improve the ability to predict VUR in patients with prenatal hydronephrosis compared with the detection rate when the trigger for VCUG is exclusively prenatal hydronephrosis.¹⁰ Applying the authors’ criteria for radiographic investigation only 97 voiding cystourethrograms were performed compared with 262 when prenatal hydronephrosis alone triggered the test. With any 1 of the 3 aforementioned findings positive on ultrasound, the positive and negative likelihood ratios

were 2.71 and 0.33, respectively. Assuming a VUR prevalence of 10% to 15% among patients with prenatal hydronephrosis, the post-test probability that a patient had VUR after a positive test (ultrasound meeting at least 1 of the 3 criteria) was 23% to 32%, and after a negative test was 6.7%.

Using their proposed criteria 11 patients who had VUR would have been missed (5 with high grade VUR and 6 with low grade VUR). However, in this subset 8 of 11 patients had VUR resolution at a mean followup of 17.3 months and only 1 of 11 had a UTI that actually occurred after VUR resolution. Since some physicians more selectively obtain VCUG only when high grade prenatal hydronephrosis is present, the authors applied this criterion to their prenatal hydronephrosis population. Using this parameter they would have performed 163 voiding cystourethrograms and would have missed a similar number of patients with VUR (10 vs 11).

The ability to more accurately predict which patients with prenatal hydronephrosis will have VUR on VCUG based on ultrasound findings is an important step in decreasing over-testing. However, we are still left with the more problematic issue of over diagnosis. For many patients with prenatally detected VUR the condition is a benign entity that often resolves spontaneously.⁵ The diagnosis of VUR in this subset of patients carries little to no clinical benefit and arguably may be detrimental.

By limiting diagnosis to those with clinically significant VUR, we could prevent not only the potential iatrogenic consequences of VCUG in clinically insignificant cases, but also the downstream effects of diagnosis such as antibiotic exposure, repeat imaging, clinic visits, and patient and parental anxiety. Central to the solution is the ability to consistently identify those patients who will have sequelae from VUR. Although specific ultrasound findings may enhance our ability to predict which patients should be evaluated with VCUG, unnecessary testing for and diagnosis of VUR will continue until we establish better markers to identify which children are at highest risk for clinically significant VUR.

To V(CUG) or not to V(CUG) is not precisely the question we should be asking. Rather, for whom do we obtain VCUG? "That is the question."¹

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