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To cite this article: Marco Zaffanello (2009) Prospective in the Medical Treatment of Reduced Renal Growth and Function by High Grade Vesicoureteral Reflux in Children, *Renal Failure*, 31:2, 178-179, DOI: [10.1080/08860220802595971](https://doi.org/10.1080/08860220802595971)

To link to this article: <https://doi.org/10.1080/08860220802595971>



Published online: 07 Jul 2009.



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## LETTER TO THE EDITOR

# Prospective in the Medical Treatment of Reduced Renal Growth and Function by High Grade Vesicoureteral Reflux in Children

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Vesicoureteric reflux (VUR) occurs when there is retrograde passing of urine back up the ureter. Urinary tract infections (UTIs) have been considered the main cause of permanent renal parenchymal damage in children with VU reflux. Until now, management of this condition in children has been directed at preventing UTIs by antibiotic prophylaxis and/or surgical correction of VUR. Therefore, Pennesi et al. reported in a recent multicenter, open label, randomized controlled trial the ineffectiveness of antibiotic prophylaxis in avoiding new renal scars in children with grade II/IV VUR. During a follow-up of four years, antimicrobial prophylaxis was unable to reduce the risk of recurring pyelonephritis and new renal scars in children compared to untreated controls.<sup>[1]</sup> However, one of our observational studies reported that surgical intervention and pharmaceutical approaches lead to similar renal outcome, quelling suspicions that today's available measures to limit renal damage are insufficient. In particular, we found that high-grade VUR was associated with kidneys that were already undersized at birth controlling/avoiding the UTIs. In addition, both early antimicrobial prophylaxis, given after prenatal detection, and a surgical approach do not seem to alter the renal outcome: refluxing kidneys always seem to grow slower than non-refluxing ones.<sup>[2]</sup> In agreement, it is uncertain whether the treatment of children with VUR confers clinically important benefits. From a recent metaanalysis, the additional benefit of surgery over antibiotics alone was reported to be minimal at best, as no concomitant reduction in risk of new or progressive renal damage was shown after 10 years.<sup>[3]</sup>

Studies published in the last years, and in particular improvements in prenatal diagnosis, have focused attention

on the association between renal dysplasia and VUR as cause of chronic renal failure.<sup>[4]</sup> On the basis of these findings, it is necessary to set new guidelines for the treatment of children with VUR, as emerging knowledge agrees with the failure of antimicrobial prophylaxis in preventing renal scars and recurrence of UTIs.<sup>[5]</sup> However, updated clinical information agrees with the suspicions that moderate to severe VUR may be associated with a prenatal renal programming, which leads to a delay in kidney growth. Obviously, when the VUR is monolateral, the contra-lateral kidney often undergoes an adjunctive overgrowth in compensation, but when the VUR involves both kidneys, the renal growth delay may be mutual.

Renal dysplasia associated with grade IV/V of VUR has its own natural progression independent of pyelonephritis recurrences. The incidence of renal failure has been stable even in years of aggressive treatment approach. Interestingly, microalbumin excretion rate in children with high-grade of VUR (IV-V) was significantly increased than those with low-grade VUR and control. In children with high-grade of VUR, both microalbuminuria and reduction of renal function may be a possible consequence of retrograde urine flow, glomerulosclerosis, and hyperfiltration.<sup>[4]</sup> Perhaps therapy with ACE-inhibitors in patients affected by proteinuria and/or hypertension could prevent the worsening of nephropathy.<sup>[6,7]</sup> but no studies about this item are yet available. At the present time, it is impossible to modify significantly the history of patients with severe bilateral nephropathy from VUR.

## DECLARATION OF INTEREST

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

Received and accepted 24 October 2008.

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