also found a declining role of calcineurin-inhibitor toxicity and concluded that most cases of kidney allograft loss have an identifiable cause.

There is, therefore, a strong need for integrating and corroborating the morphological study of kidney allograft biopsies with the newly emerging technologies such as molecular genetic, omics, and donor-specific antibody studies to better identify the "specific disease phenotypes" of chronic allograft injury, which can perhaps, then, translate into better and personalized management of kidney transplant recipients and better long-term graft outcomes.[4] In our view, this integration is all the more important and relevant in the context of IFTA than in the setting of acute kidney dysfunction.

Muhammed Mubarak

Department of Histopathology, Sindh Institute of Urology and Transplantation (SIUT), Karachi, Pakistan
E-mail: drmubaraksiut@yahoo.com

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Re: Usefulness of Serum Procalcitonin Level for Prediction of Vesicoureteral Reflux in Pediatric Urinary Tract İnfection

Dear Editor,

We have read with interest the recently published article entitled "Usefulness of serum procalcitonin level for prediction of vesicoureteral reflux in pediatric urinary tract infection" by Mortazavi and Ghojazadeh.[1] They aimed to evaluate the predictive value of procalcitonin in describing vesicoureteral reflux (VUR). They concluded that elevated procalcitonin level may be used for prediction of all grades of VUR in children with febrile urinary tract infection. We would like to thank the authors for their contribution.

Procalcitonin, a 116-amino acid propeptide of calcitonin, is synthesized by the parafollicular C cells of the thyroid and involved in calcium homeostasis. [2] Several studies have demonstrated that procalcitonin levels rise in inflammatory states following bacterial or fungal infections, tumors, trauma and surgery. Procalcitonin can be a useful tool for diagnosis of sepsis and it can be used as a guide for antibiotic therapy in individual patients as a surrogate biomarker.

Urinary tract infections are crucial in young children since they can lead to serious problems such as kidney infections, permanent renal damages and end-stage renal failure.VUR is one of the most important predisposing factors of urinary tract infections in children and if it is left untreated, it can cause renal tissue inflammation and kidney damages.[1] Recently, procalcitonin has been proposed as a novel biomarker for prediction of VUR.[1]

Nonspecific elevations in procalcitonin levels can typically be seen in situations such as massive

stress, although there is no bacterial infection. In these situations, after the elevation of procalcitonin values, a rapid decline is observed in the follow-up measurements. [3] On the other hand, procalcitonin can have false negative or lower values in early course and localized site of an infection. [3] In this context, repeated measurements of procalcitonin could have been performed. Therefore, it would have been better if the authors had mentioned these conditions as limitations.

Lastly, procalcitonin is affected by a variety of infectious agents. Microbiological assessment is crucial while evaluating the procalcitonin levels. In comparisons of Gram negative agents with Gram positives, procalcitonin levels have been found to be higher in Gram negatives. [4] Therefore, it would have been more accurate, if the authors had evaluated procalcitonin levels according to infectious agents in greater detail in this study.

In conclusion, further studies are needed to determine the association between procalcitonin and VUR. We are of the opinion that procalcitonin should be evaluated with other independent variables and markers (eg, bacterial agents, C-reactive protein, and erythrocyte sedimentation rate) to provide the required information about the inflammatory status of the patient.

Ergenekon Karagoz,* Asim Ulcay

Department of Infectious Diseases and Clinical Microbiology, GATA Haydarpasa Training Hospital, Istanbul, Turkey
E-mail: ergenekonkaragoz@hotmail.com

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Re: Kartagener Syndrome With Focal Segmental Glomerulosclerosis

Dear Editor,

The article published by Momeni and colleagues, entitled "Kartagener syndrome with focal segmental glomerulosclerosis" in the esteemed *Iranian Journal of Kidney Diseases* had some interesting points.[1] In this article, they explained a 27-year-old female patient as a case of Kartagener syndrome. Renal biopsy in this case showed segmental scar with adhesion to the Bowman capsule, which was indicative of focal segmental glomerulosclerosis (FSGS).[1] We would like to remind a few points. Indeed, it is indispensable to classify the variant of FSGS, too. In 2004, a group of renal pathologists suggested a consensus-based morphological classification system for FSGS based exclusively on light microscopic examination, commonly known as the Columbia classification.[2-5] According to this classification, 5 morphologic variants of FSGS are described: FSGS, not otherwise specified (NOS) or the classic type; cellular variant; collapsing variant; perihilar variant; and tip variant.[5-7] In brief, tip variant of FSGS necessitates the exclusion of collapsing variant and presence of at least one glomerulus with segmental lesion involving the tip domain of the glomerular capillary tuft. In the perihilar variant, the segmental sclerotic lesion is situated at the vascular pole and requires the exclusion of collapsing, tip, or cellular lesion. The cellular variant needs exclusion of collapsing and tip lesions, and is defined by segmental endocapillary hypercellularity occluding lumina in at least 1 glomerulus.[5-8] Collapsing variant was defined by collapse of at least 1 capillary loop with hyperplasia and hypertrophy of overlying visceral epithelial cells, irrespective of the presence of other variants of FSGS. In cases where none of these definitions