# Re: Hyperlipidemia After Kidney Transplantation: Long-term Graft Outcome

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Dear Editor,

We read with interest the article by Raees-Jalali and colleagues[1] recently published in the *Iranian Journal of Kidney Diseases*, titled "Hyperlipidemia after kidney transplantation: long-term graft outcome." They evaluated the prevalence of hyperlipidemia and its relation with renal allograft function within 10 years of follow-up among 73 kidney transplant patients in a single center. They reported that hypercholesterolemia in the first year after transplantation was only associated risk factor for renal allograft impairment.

We agree that the kidney allograft dysfunction can play a role in post-transplant hyperlipidemia; however, causes of dyslipidemia after renal transplantation are usually multifactorial, especially the type of immunosuppression regimens and genetic predisposition missed in this study. In a retrospective cross sectional study performed on 1391 kidney transplant recipients, we found that dyslipidemia had correlation with age of recipient, serum creatinine and cyclosporine blood levels (unpublished data). In logistic regression, however, serum creatinine was only risk factor for hypercholesterolemia development after kidney transplantation (odds ratio, 1.6; 95% confidence interval, 1.4 to 1.8). In our previous study on 687 kidney transplant recipients, there was no significant correlation between graft survival and severity of hyperlipidemia.[2]

In the study of Raees-Jalali and coworkers,[1] deceased-donor kidney transplantation was done in 22% of all patients. They did not compare the rate of dyslipidemia between living and deceased kidney recipients. In our study, hypercholesterolemia and low high-density lipoprotein cholesterol were significantly more detected in deceased kidney recipients when compared to living kidney recipients (unpublished data); thus, ischemia could induce dyslipidemia in the future. There is one study about this variable.[3]

Raees-Jalali and coworkers[1] noted that the incidence of dyslipidemia decreased over the time after kidney transplantation with peak incidence of altered lipid profile in the first year after transplantation. We observed that the cholesterol and triglyceride levels were significantly higher 4 to 12 months after transplantation than their level in the first 3 months and beyond 1 year after kidney transplantation.

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# Re: Pediatric Urolithiasis: an Experience of a Single Center

Dear Editor,

We read with interest an article recently published in the Iranian Journal of Kidney Diseases, entitled "Pediatric urolithiasis an experience of a single center" by Safaei Asl and Maleknejad.[1] In this retrospective study performed on 84 children with

kidney calculi, the clinical features and risk factors of urolithiasis were evaluated.[1] They concluded that urolithiasis remains a serious problem in Iranian children. In addition, family history of urolithiasis, urologic abnormalities, metabolic disorders, and urinary tract infections were associated risk factors for pediatric urolithiasis.[1]

We agree that the wide geographic variations in pediatric urolithiasis exist in terms of the incidence of lithiasis in childhood, site of formation, stone composition, and predisposing etiological factors. Authors noted that the stones were only located in 90.6% of the cases in the upper urinary tract and in 2.4% it was only in the bladder[1]; however, in a Tunisian study on 300 children with urolithiasis, the calculi were located in the kidneys in 69.0% of patients and bladder stones were observed in 27.7% of cases.[2] Importantly, bladder stones have been reported to be endemic in Asia such as Tunisia.[2] Infants appear to be more affected by bladder stones than teenagers (42.4% versus 16.1%, $P < .001$).[2] Family history of renal stones was noted in 8% of Tunisian cases,[2] while it was reported in 27% of Iranian pediatric patients.[1] A history of urinary tract infection was the same in the both studies, approximately 25% of individuals.[1,2] Metabolic disorders were reported in 53% of Iranian pediatric with urinary calculi,[1] while it was noted in 9% of Tunisian patients.[2] In both studies, urolithiasis was more likely to be occurred in boys than girls, and the male-female ratio was 1.4:1 in Iranian cases and 1.54:1 in Tunisian subjects.[1,2] Furthermore, in most studies male predominance was observed among pediatric urolithiasis.[3,4] The urological abnormalities in Iranian children was also similar to Tunisian patients (14% versus 11%),[1,2] which matches with other studies.[5,6]

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# Re: Multiple Myeloma Presenting as Acute Tubulointerstitial Nephritis and Normal Serum Protein Electrophoresis

Dear Editor,

In the Volume 6, Number 1, January 2012 issue of the *Iranian Journal of Kidney Diseases*, there was an interesting case report with the title of "Multiple Myeloma Presenting as Acute Tubulointerstitial Nephritis and Normal Serum Protein Electrophoresis" written by Momeni and colleagues.[1] They presented a case of multiple myeloma undergone renal biopsy that light microscopic study of the prepared slides showed necrotic materials, shed tubular cells, and hyaline casts in the tubule lumens and 30% tubular atrophy. Most parts of the interstitium were occupied by lymphocytic infiltration, some of them invaded to tubules and then they diagnosed acute tubulointerstitial nephritis in association with some degrees of chronicity of the interstitial area based on these findings. The pictures of renal biopsy showed some polymorphonucleocytes in tubular lumen and a few dense small casts.

Finding polymorphonucleocyte aggregations in the tubular lumina is more in favor of an acute pyelonephritis rather than acute tubulointerstitial nephritis. Although in a patient with multiple