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

myeloma finding polymorphonucleocytes in the tubules surrounding the irregular large casts is a usual feature of myeloma cast nephropathy or light chain cast nephropathy (LCCN), but sometimes in a few conditions, polymorphonucleocyte aggregation in tubular lumens is the prominent finding, and in these cases, instead of diagnosing an acute bacterial pyelonephritis or acute tubulointerstitial nephritis, full search of the slides (even preparing more sections) for finding the light chain casts, especially in distal tubules and collecting tubules, will lead us to a diagnosis of LCCN. Chronic tubulointerstitial nephritis is a usual finding in LCCN as this case showed in the first and second biopsy specimen.[2]

I would suggest the authors to have more sections of the first specimen, looking for irregular dense eosinophilic casts, especially in distal tubules with peripheral cell reaction as this case seems to be a usual case of multiple myeloma with LCCN. Although the renal biopsy findings are correlated with the paraclinical findings of presence of kappa light chain in urine as was mentioned in the article.

Unless there is rapid intervention for LCCN, including effective chemotherapy regimens to reduce light chain production and emerging extracorporeal techniques to remove circulating light chains, progressive and irreversible damage occurs, particularly interstitial fibrosis and tubular atrophy as this case showed in the second biopsy.[3,4]

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