

Obstructive Uropathy as Cause of Rickets: Often A Forgotten Entity

Sir,

In India, not frequently, children with leg deformity and growth retardation are referred to nephrologists in late stages. Many of them, by then had undergone unsuccessful surgical treatment for straightening of legs, undergone unnecessary extensive investigations and received treatment for dwarfism and for proximal myopathy. Though its incidence is unknown and literature is scanty on the subject, children suffering from rickets may have obstructive uropathy as the underlying cause as our study revealed. If diagnosed early these children can be managed better.

Between the year 1990 and 2001, 37 children (age 7-14 yr) with leg deformity were investigated. Vesicoureteric reflux (59.4%), pelvi-ureteric junction obstruction (10.8%) and bladder neck obstruction (8.1%) were detected in these cases. Primary renal tubular acidosis was found in 21.6%. Obstructive uropathy with normal BUN and serum creatinine (S.Cr: 1.2 ± 0.45 mg/dL) constituting 44.8%, showed laboratory feature of distal renal tubular acidosis (RTA); while those (55.2%) with modest rise in BUN and serum creatinine (S.Cr: 1.9 ± 0.65 mg/L) showed in addition to RTA, elevated parathormone (68.7%) and calcium and phosphorus abnormalities (56.2%) respectively. Although serum vitamin-D level was inconclusive in the former group (44 ± 9.5 ng/ml (range 24-40)), it was low in 75.6% of patients in the latter group (27 ± 4.2 ng/ml (range 24-40)). Surgical relief of obstruction and treatment of RTA resulted in clinical and radiological improvement in 31.0 of patients.

Long standing obstructive uropathy in children can

cause renal tubular dysfunction manifesting as acidosis long before glomerular dysfunction (azotaemia).¹ This probably explains late referral to renal services. Our data suggests RTA as the cause of rickets. Renal insufficiency developed later in some later in some patients resulting in additional problem of disturbances in calcium, phosphorous and vitamin-D, which may have contributed to clinical manifestations in this group. Secondly nutritional deficiency of vitamin-D and protein may have aggravated rickets in growing children belonging to the poorer socio-economic class. This probably explains why we see such cases in India, but is reported rarely in richer countries.² Nevertheless a general lack of awareness of nonazotemic renal bone disease in children due to remediable cause like obstructive uropathy does exist.

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