

## Tongue Biting and Epilepsy

I read with interest the recent report describing the requirement of partial glossectomy for lingual edema following an epileptic fit(1). I would like to share my thoughts on the importance of tongue biting in epilepsy.

Tongue biting, traditionally believed to be a specific clinical sign favoring epilepsy, is also reported to occur in syncope and psychogenic seizures(2). Lateral tongue biting (on the sides) is more specific for a diagnosis of true epilepsy as compared to syncope(2) or psychogenic seizures, where biting of the lip or tip of the tongue is seen(3). Nocturnal tongue biting (NTB) can rarely be the sole manifestation of epilepsy(4) and typically occurs in frontal lobe seizures.

In a recent study, 53% of those interviewed mentioned that they would put an object in patient's mouth to prevent tongue biting (33% learned this from a local television program)(5). Therefore, educating the patient's caregivers about methods of

preventing tongue biting should form part of any effective epilepsy management.

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## Nephrocalcinosis in a Child with Nephrotic Syndrome

We report an uncommon association of nephrotic syndrome with normocalcaemic borderline hypercalciuria and early nephrocalcinosis detected on a renal biopsy.

An eighteen-month-old male toddler presented with onset of nephrotic syndrome at

eleven months of age. This had been adequately treated with six weeks of daily followed by six weeks of alternate day steroids with a good response. He relapsed within two months of stopping steroids. During the second episode there was no response to full dose steroids given for four weeks. The child was hepatitis B surface antigen negative and no focus of infection was detected as a cause for secondary resistance. He also had noted hypertension and

microscopic hematuria. He was being treated with enalapril and frusemide and steroids for four weeks prior to hospitalization.

The child was severely cushingoid and hypertensive with proximal muscle weakness in the lower limbs. Investigations revealed 3+ albuminuria with urine protein/creatinine ratio  $>2$ , serum albumin 2.3 g/dL and serum cholesterol 256 mg/dL. Renal function tests were normal. The venous blood gas study and serum electrolytes were normal with no evidence of metabolic acidosis or alkalosis ruling out the possibility of a coexisting tubulopathy. Serum calcium was 9.5 mg/dL, phosphorus 4.2 mg/dL and alkaline phosphatase 225 IU/L. The urinary spot calcium/creatinine ratio was 0.42 (normal at 18 m-6 y  $<0.42$ ) and 24-hr urinary calcium excretion 54 mg (6 mg/kg) with no evidence of nephrocalcinosis on plain X-ray KUB or renal ultrasound. A kidney biopsy revealed mesangial proliferative glomerulonephritis with tubules showing significant calcification suggestive of nephrocalcinosis.

Nephrocalcinosis has been reported uncommonly in association with nephrotic syndrome(1). The more common causes for nephrocalcinosis include prolonged diuretic therapy with frusemide, hypervitaminosis D, distal renal tubular acidosis, prolonged immobilization, cushing syndrome, hyperoxaluria, hyperuricosuria, hyperparathyroidism and hypophosphatemic rickets(2). Nephrocalcinosis in preterm babies following frusemide therapy has been well documented(3). Evaluation and treatment

aims at determining the presence of hypercalciuria.

Our patient was treated with three doses of pulse methylprednisolone therapy followed by oral alternate day prednisolone in tapering doses and cyclophosphamide. In view of his nephrocalcinosis with borderline hypercalciuria, frusemide was stopped. A combination of thiazides and spironolactone was used to control edema in conjunction with restriction of salt intake. Repeat urine Ca/Cr ratio was 0.18.

One should be aware of nephrocalcinosis as a side effect of prolonged diuretic therapy and periodically monitor urine for hypercalciuria in children on such therapy.

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