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.

Sudhir Kumar.

Neurology Unit, Department of Neurological Sciences, Christian Medical College, Vellore, Tamilnadu 632 004, India. E-mail: drsudhirkumar@yahoo.com

REFERENCES

- Singh K. Partial glossectomy for lingual edema following injury. Indian Pediatr. 2004; 41: 520-521.
- Benbadis SR, Wolgamuth BR, Goren H, Brener S, Fouad-Tarazi F. Value of tongue biting in the diagnosis of seizures. Arch Intern Med. 1995; 155: 2346-2349.
- De Toledo JC, Ramsay RE. Patterns of involvement of facial muscles during epileptic and non-epileptic events: review of 654 events. Neurology. 1996; 47: 621-625.
- Vasiknanonte P, Kuasirikul S, Vasiknanonte S. Two faces of nocturnal tongue biting. J Med Assoc Thai. 1997; 80: 500-507.
- Fong CY, Hung A. Public awareness, attitude, and understanding of epilepsy in Hong Kong Special Administrative Region, China. Epilepsia. 2002; 43: 311-316.

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