Primary hyperparathyroidism in a child

Sir,

I have two comments on the interesting case report by Anitha *et al*.[1]

First, Anitha *et al*. mentioned that in the view of diagnosing parathyroid adenoma by suggestive clinical presentation, laboratory investigations, and imaging studies in their studied patient with primary hyperparathyroidism (PHT), the patient underwent left upper parathyroidectomy under peroperative radio-guidance. As a part of management, the patient was kept under regular clinical and laboratory follow-up to ensure complete recovery. Postoperatively, serum calcium reduced to a nadir of 7.0 mg/dl, and the patient was treated with intravenous calcium gluconate initially followed by oral calcium and Vitamin D. Three months after the surgery, serum calcium was 10.1 mg/dl. However, the immediate pre- and post-surgery parathyroid hormones (PTHs) were 1619 pg/ml and 203 pg/ml respectively (normal range = 11–67 pg/ml). The authors did not attempt to make serial PTHs estimations during follow-up to determine whether that postoperative PTH elevation was transient or persistent. In addition, the authors relied upon clinical follow-up with regressions of symptoms to determine recovery. The persistently elevated postoperative PTH in the case in question is worrisome. It is worth mentioning that late elevation in PTH after successful surgery occur in 21.5% of cases. Multiple causes have been implicated, including reactive hyperparathyroidism, hungry bone syndrome, Vitamin D deficiency, renal dysfunction, and ethnic or lifestyle differences.[2] In mild observation of the patient may be sufficient. In cases of reactive hyperparathyroidism due to hypocalcemia, administration of calcium is indicated and in symptomatic patients, additional administration of Vitamin D or calcitriol is necessary. In cases of ongoing hyperparathyrinemia, an interdisciplinary diagnostic and therapeutic approach is required.[2]

Second, multiple endocrine neoplasia type 2 (MEN2) is an autosomal-dominant cancer syndrome characterized by variable penetrance of medullary thyroid carcinoma, pheochromocytoma, and PHP. It consists of two clinical subtypes, MEN2A, and MEN2B. Familial medullary thyroid cancer is now viewed as a phenotypic variant of MEN2A with decreased penetrance for pheochromocytoma and PHP rather than a distinct entity. Genotype-phenotype correlations exist that help to predict the presence of other associated endocrine neoplasms as well as the timing of early thyroid carcinoma development.[3] It has been noticed that PHP occurs in 10–30% of patients with MEN2A and rarely as the sole clinical manifestation.[4] A germline mutations in the RET proto-oncogene is responsible for the MEN2 syndrome, particularly mutations in the codon 634 and that in the majority of MEN2 cases, a germline RET mutations can be identified by genetic testing.[3] Genetic screening for these mutations has been extensively exploited worldwide to optimize the diagnostic and clinical management of MEN2 patients and their relatives and offers the opportunity to regular monitoring of potential malignancies and timely intervention for these patients like early prophylactic thyroidectomy.[5] Genetic screening ought to be considered in the case in questions and patient's relatives. However, the limited financial resources in India have probably curbed Anitha *et al*.[1] from arranging it.

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