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PARATHYROID ADENOMA: CASE REPORT

Iv. Rachkov[1], A. Julianov[1], H. Hristov[1], A. Karashmalakov[1], Y. Georgiev[1*], A. Zdraveski[1], H. Stoyanov[1], Sl. Paliiska[1], M. Karamalakov[1], B. Belchev[1], G. Ganchev[1], T. Mircheva[2]

[1]Surgery Department, Medical Faculty, Trakia Universtity, Stara Zagora, Bulgaria
[2]Department of Endocrinology, Medical Faculty, Trakia Universtity, Stara Zagora, Bulgaria

ABSTRACT

Parathyroid adenoma is part of a spectrum of parathyroid proliferative disorders that includes parathyroid hyperplasia, parathyroid adenoma, and parathyroid carcinoma. Patients typically present with evidence of primary hyperparathyroidism with elevated serum calcium levels and elevated serum parathyroid hormone levels.

Sonography and 99mTc-sestamibi scintigraphy are the primary imaging modalities utilized for the visualization of diseased glands. Histological examination alone can't distinguish hyperplasia from adenoma.

We present a case of parathyroid adenoma in a patient with previous medical history of nephrocalcinosis, mild chronic renal failure, and surgically extirpated osteoclastoma on the left forearm. Preoperative work-up revealed increased levels of calcium and parathyroid hormone, together with tumor mass in the lower right lobe of the thyroid with increased activity on 99mTc-MiBi scintigraphy.

After operative treatment calcium and parathyroid hormone levels returned to normal ranges.

Parathyroid adenoma should always be suspected in patients with renal failure, together with bone tumors.

Key words: parathyroid hyperplasia, preoperative work-up, elevated serum calcium

INTRODUCTION

In 1800 Ivar Sanstrom, a Swedish medical student, first described the human parathyroid glands. It was not recognized until 1815, 15 years later, that excision of the parathyroid glands, rather than the removal of the thyroid, was the cause of tetany. In 1907 Halsted commented, 'it seems hardly credible that the loss of bodies so tiny should be followed by a result so disastrous'

Parathyroid adenoma is part of a spectrum of parathyroid proliferative disorder that includes parathyroid hyperplasia, parathyroid adenoma, and parathyroid carcinoma. Patients typically present with evidence of primary hyperparathyroidism with elevated serum calcium levels and elevated serum parathyroid hormone levels. Hyperparathyroidism is divided into primary, secondary, and tertiary hyperparathyroidism. Most parathyroid hyperplasia is the result of secondary hyperparathyroidism due to renal disease. Tertiary hyperparathyroidism is the autonomous secretion of parathyroid hormone in the setting of long-standing renal disease resulting in hypercalcemia. Eighty to 85 percent of primary hyperparathyroidism is caused by parathyroid adenoma followed by primary parathyroid hyperplasia (15%) and parathyroid carcinoma (5%).

Patients with primary hyperparathyroidism may present with clinical evidence of elevated serum calcium levels which include non-specific symptoms such as fatigue, pain and weakness as well as polydipsia, polyuria, and nephrolithiasis. Gastrointestinal symptoms

***Correspondence to**: *Yonko Georgiev, MD, Surgery Department, Trakia Universtity, 11 Armeiska str, Stara Zagora, Bulgaria, Tel: 0899 127 849, e-mail: yonko_georgiev@abv.bg*

include constipation, anorexia, nausea, and vomiting. Extreme hypercalcemia can lead to cardiac arrhythmias, coma and death. These days most patients with hypercalcemia are discovered incidentally on routine work-up for other reasons. Elevated levels of serum calcium are noted on laboratory screening tests. Evaluation consists of radiographic studies and subsequent surgical exploration.

CASE REPORT

A 56 year old caucasian male was admitted to the surgery department with palpable tumor mass on the right side of the neck. His previous medical history included nephrocalcinosis, chronic renal failure 1st degree, and surgically extirpated tumor on the left forearm, which pathological examination showed to be osteoclastoma. Laboratory data revealed extremely elevated parathyroid hormone- 2500 pg/ml, elevated calcium levels – 3,35mmol/l (total) and 1,58mmol/l (ionized), elevated blood urea nitrogen- 14.09mmol/l and creatinine- 138 mmol/l, together with hemoglobin 109 g/l, the rest of the blood test was within the normal ranges. Ultrasound imaging showed tumor formation 5cm in diameter in the right lower lobe of the thyroid **(fig. 1)**. Further examination of the patient included 99mTc-MiBi scintigraphy which displayed an area of increased activity in the lower right lobe of the thyroid gland **(fig. 2)**. With preoperative diagnosis parathyroid adenoma patient was scheduled for operation.

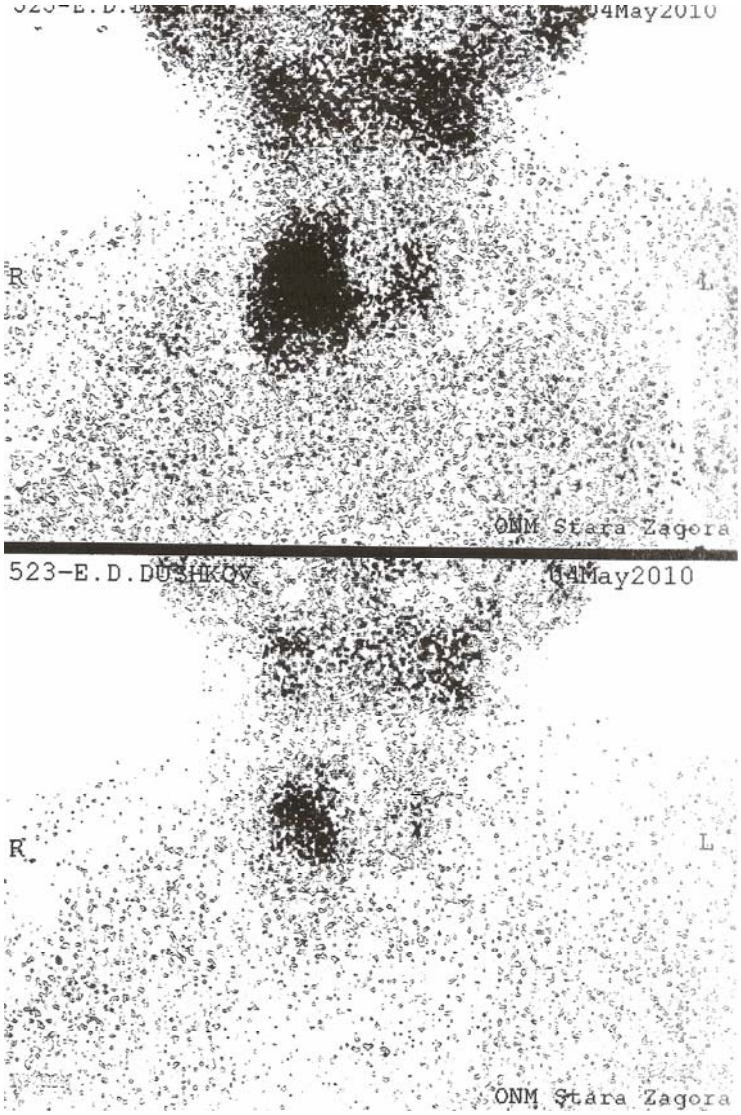

Fig. 1. Preoperative Scintigraphy

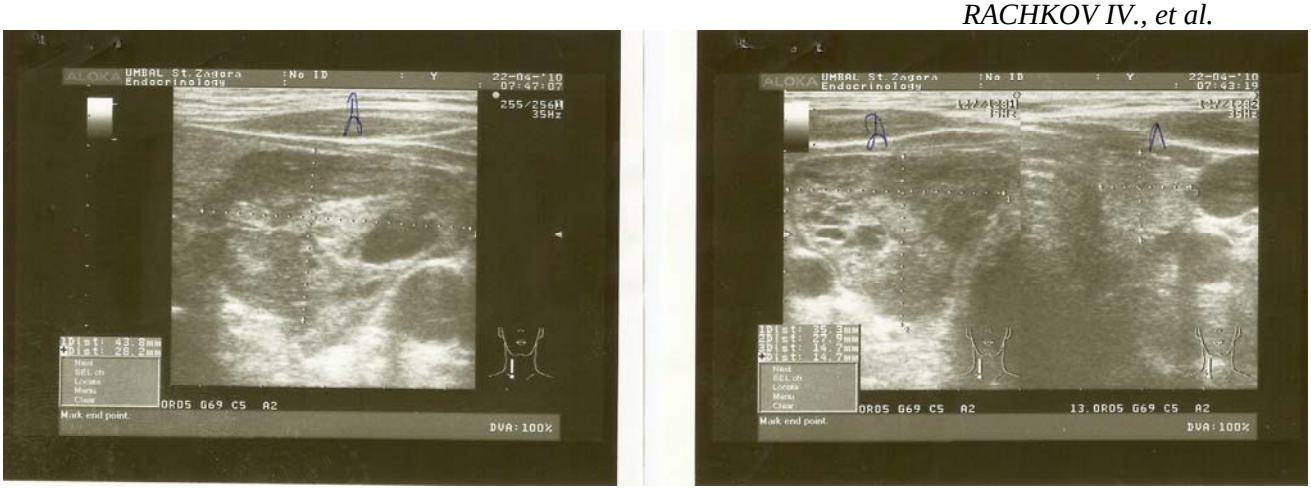

Fig. 2. Preoperative US

Using a standard Kocher incision the lower right pole of the thyroid gland was reached. The tumor was found originating from the lower right parathyroid gland and macroscopically there was no infiltration to surrounding structures. The formation was carefully dissected and excised **(fig. 3, 4 and 5**). Frozen section confirmed the diagnosis: parathyroid adenoma.

Postoperative period was uneventful. Laboratory test showed calcium levels within normal ranges. Levels of parathyroid hormone decreased to normal range. One year after the operation the patient has no complaints.

DISCUSSION

Normal parathyroid glands are too small to be detected on imaging (usually 5x4x1 mm), but parathyroid disease typically results in enlargement of the glands allowing for visualization. Sonography and 99mTc-sestamibi scintigraphy are the primary imaging modalities utilized for the visualization of diseased glands [1]. Scintigraphy is approximately 90% sensitive for localizing a parathyroid adenoma, and can easily demonstrate glands greater than 500 mg[2]. Ultrasound imaging demonstrates parathyroid adenomas as typically homogeneously hypoechoic lesions compared with the adjacent thyroid, and they can easily be detected when they are larger than 1 cm. Cystic parathyroid adenomas are rare, and the cystic areas appear as regions of decreased echogenicity within the gland. Doppler imaging typically demonstrates a characteristic extrathyroidal feeding vessel entering the parathyroid gland at one of the poles. Contrast enhanced CT and MRI are less commonly used for preoperative localization, but may be of benefit in the setting of localization of ectopic glands.

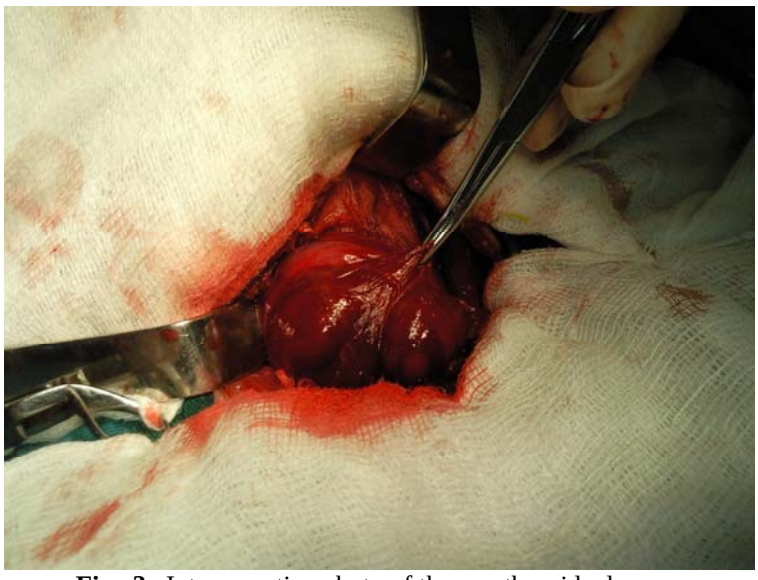

Fig. 3. Intraoperative photo of the parathyroid adenoma

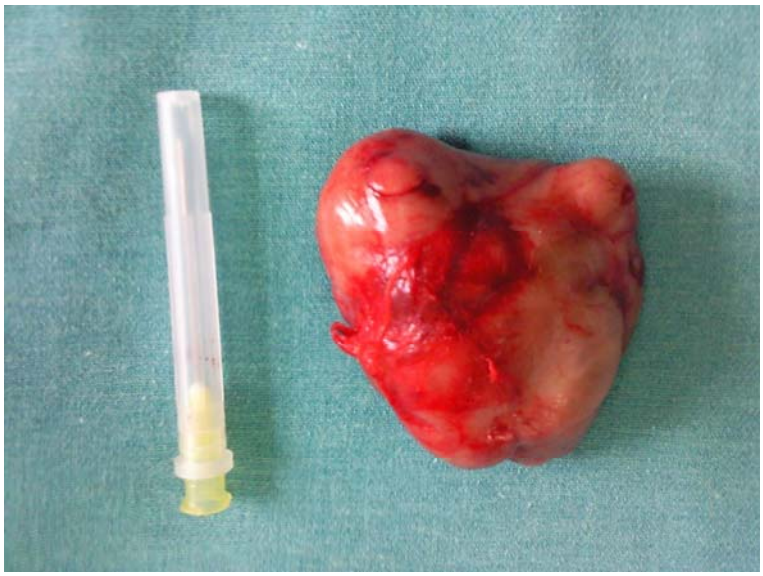

Fig. 4. The resected specimen

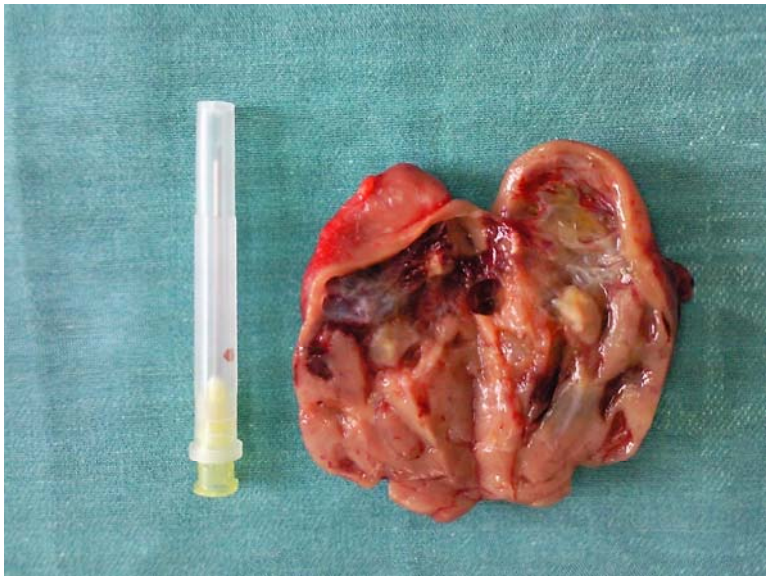

Fig. 5. The resected specimen

Parathyroid hyperplasia typically involves all four glands, i.e. all four glands are enlarged. Occasionally parathyroid hyperplasia may be uneven and one or two glands are more prominent and enlarged than the other glands causing confusion radiographically and on clinical examination. If one gland is significantly larger than the other glands it may be interpreted as parathyroid adenoma rather than hyperplasia and following resection of the presumed ‘‘adenoma’’ the hyperparathyroidism does not resolve. So how these two conditions could be distinguished? None of the histological features said to distinguish hyperplasia from adenoma have survived after evaluation of large numbers of cases. Historically, the distinction was guided by the doctrine that an encapsulated mass compressing adjacent parathyroid tissue defined an adenoma. This has not proved reliable; some adenomas lack discernable capsules, and some hyperplasias have a nodular nature, compressing surrounding parathyroid tissue [3, 4]. From the clinical point of view, only one guide has withstood the test of time: removal of only the enlarged glands. This approach has produced a high rate of cure and a very low rate of postoperative hypoparathyroidism [5].

Patients with MEN syndromes, most commonly MEN 1, frequently will have parathyroid proliferative disorder as part of their syndrome. Nearly 90% of patients with

MEN 1 have parathyroid hyperplasia. Parathyroid adenoma and carcinoma can also be seen as part of these syndromes. While the vast majority of patients with parathyroid proliferative disorder present with sporadic disease, the possibility of an MEN syndrome should always be kept in mind when evaluating these patients.

Presentation of hypercalcemia and normal PTH level due to parathyroid adenoma is infrequent; however, its incidence has been estimated to be between 5% and 33%. Since 1976, approximately 140 cases of hypercalcemia and normal PTH levels due to parathyroid adenoma have been reported in the literature. However, no correlation between the atypical biochemical presentation, histological features and causative factors has been demonstrated[6-9]. Hollemberg [7] and Perez [8] proposed the following theoretical explanations to the mechanism of inappropriately low PTH level in the PHPT cases observed: 1/a circulating inhibitor of PTH;2/ the pulsatile secretion of the hormone; 3/an abnormal PTH molecule with increased biologic activity;4/ increased peripheral tissue sensitivity to normal PTH; or 5/the presence of another mediator of hypercalcemia. None of these hypotheses have been verified.

Finally, parathyroid adenoma should always be suspected in patients with renal failure, together with bone tumors.

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