ORIGINAL ARTICLE

PRESENTATION AND MANAGEMENT OF PARATHYROID TUMORS

Wajahat Hussain Wasty , Khalid Hussain Mahida , Khalid Iqbal

ABSTRACT

Objective: To evaluate cases presenting with parathyroid tumors and the efficacy of treatments offered.

Methods: Six cases presented with bony manifestations in mandible, femur, tibia and maxilla while one presented with an overt neck swelling. Renal calculi showed in one case. All cases were subjected to assessment of serum parathyroid hormone level and radionuleotide scan.Sonography was done in addition in two of the cases . In all cases parathyroid tumors were identified and subjected to exploratory parathyroid surgery, In addition bony pathologies were subjected to excisions of the lesions.

Results: In all the cases serum parathyroid hormone levels were raised.MIBI scans picked parathyroid tumors in almost all the cases. Six of the excised tumors were parathyroid adenomas while one was adenocarcinoma. Additional surgeries done in these cases include excisions of mandibular growths, maxillectomy and osteotomies on tibia and femur. All were giant cell tumours.One case with adenocarcinoma expired. The other cases showed normalization of parathyroid hormone levels.

Conclusion: Parathyroid adenoma is the common benign tumor of parathyroid gland usually presenting with exaggerated parathyroid hormone levels. Ultrasound scan screening with tc99 Sestamibi scanning are most helpful in establishing the diagnosis. Parathyroid surgery is safe in experienced hands and is the standard treatment modality to reverse the morbidity associated with pathologies. In all bony lesions with a doubtful clinical diagnosis screening for Parathyroid lesions is mandatory. Gross disfigurements, functional disabilities call for excision of bony lesions.

KEY WORDS: Parathyroid Gland, Adenoma, Gaint Cell Tumour, Sestamibi Scan.

INTRODUCTION

Parathyroid glands were first described in humans in 1880; Von Recklinghausen noted the relationship of parathyroid glands to fibrocystic bone disease in 1891 with the description of von Recklinghausen disease. Parathyroid disease usually manifest itself in terms of an over functioning state called hyperparathyroidism.

Hyperparathyroidism is a condition caused by excessive and uncontrolled secretion of PTH by the parathyroid glands. Increased levels of parathyroid hormone(PTH) affect bone, the GI tract, and the kidneys, which cause elevation of the serum calcium level, generalized bone disease, decreased serum phosphorus levels. This occurs due to increased renal excretion and decreased reabsorption of calcium, and increased excretion of phosphorus.

A variety of systemic conditions that involve various tissues result from changes in serum calcium and phosphorus levels. Skin, tendons, muscles, soft tissue, kidneys, eyes, nervous system, gut, and vascular systems can be involved. This study is based on the cases picked with hyperparathyroidism and its various manifestations and the management adopted in a tertiary care setting in Karachi, Pakistan.

METHODOLOGY

Four females and 3 males reported during the study period. The age ranged between 14 and 47 years. 4 of the cases had bony manifestations in terms of giant cell and Brown tumors involving mandible, maxilla, tibia and femur. Some cases showed concurrently presenting bony lesions . In majority of the cases serum calcium was within the normal limits while the serum parathyroid hormone (PTH) was exaggerated with range of 287 to 882 pg/ml (Table 1). In one case renal calculus was seen as a major manifestation. One case was distinct where the parathyroid tumor presented as a bulging lesion in the neck close to manubrium.

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Table 1. Patient Data and Investigations

No	Name	Age	Gender	Calc	PTH	Site of Lesions	MIBI	Sonography	Tetrafosmin
1	FAS	39	Male	11.02	884.2	Neck Lesion	Positive	Not Done	Not Done
2	JM	40	Female	10.97	497.8	Femur/Kidney	Negative	Not Done	Positive
3	RG	14	Female	9.90	678.0	Tibia/Maxilla	Positive	Positive	Not Done
4	SA	32	Male	10.7	367. 0	Urolithiasis	Not Done	Positive	Positive
5	MI	47	Male	11.0	287.0	Tibia	Positive	Not Done	Not Done
6	SB	27	Female	10.7	255.0	Mandible	Positive	Not Done	Not Done
7	W	21	Female	10.5	466	Mandible	Positive	Not Done	Not Done

RESULTS

The parathyroid tumors were suspected in all these cases on the basis of exaggerated PTH hormone levels .These cases were subjected to tc99 MIBI scan and t99 tetraformin scans which picked up parathyroid tumors successfully in all but one case . In two of the cases ultrasound supported the findings. In 4 of the cases left lower parathyroid gland was involved while in two left upper gland was involved; In one case right lower gland was involved. Surgical exploration in the cases conformed to the investigative findings. One case on histopathology turned out to be parathyroid adenocarcinoma while all the other cases were parathyroid adenomata. Additional surgeries done in these cases include excision of mandibular growths, maxillectomy and osteotomies on tibia and femur. In one case of adenocarcima the patient expired on the fourth post-operative day while in all the other cases serum PTH normalized (Table 2).

Table 2. Pathologies, treatment/s offered and the results

No	Name	Pathology	Treatment	Results
1	FAS	Lower parathroid adenocarcinoma	Excision	Expired
2	JM	L upper parathyroid adenoma Femoral neck growth	Excision of PT tumor Femoral growth	Normalized
3	RG	L lower parathyroid adenoma Growth maxilla/tibia	Excision of PT tumor Maxillectomy/tibia	Normalized
4	SA	l lower Parathyroid adenoma	Excision of PT tumor	Normalized
5	MI	L upper parathyroid adenoma Growth Tibia	Excision of PT tumor Tibial growth	Normalized
6	SB	R upper parathyroid adenoma/ Mandibular growth	Excision of PT tumor mandibular growth	Normalized
7	W	L lower parathyroid adenoma/ Mandibular growth	Excision of PT tumor mandibular growth	Normalized

DISCUSSION

Primary hyperparathyroidism (PHPT) is a disorder of the parathyroid glands, also called parathyroid. "Primary" means this disorder originates in the parathyroid: One or more gets enlarged, Overactive parathyroid glands secretes excessive parathyroid hormone (PTH). In secondary hyperparathyroidism, a problem elsewhere causes the parathyroid to become overactive .This may happen in case of any renal disease or failure.

If the parathyroid glands secrete too much hormone, as happens in primary hyperparathyroidism, the balance gets disrupted: Blood calcium raises. This condition of excessive calcium in the blood, called hypercalcemia, is what usually signals that something may be wrong with the parathyroid glands. In 85 percent of people with primary hyperparathyroidism, a benign tumor called an adenoma is formed on one of the parathyroid glands, causing it to become overactive. Benign tumors are noncancerous. In most other cases, the excess hormone comes from two or more enlarged parathyroid glands, a condition called

l hyperplasia. Very rarely, hyperparathyroidism is caused by cancer of a parathyroid gland. [1]

This excess PTH triggers the release of too much calcium into the bloodstream. The bones may lose calcium, and additional calcium may be absorbed from food. The levels of calcium may increase in the urine, causing kidney stones. PTH also lowers blood phosphorus levels by increasing excretion of phosphorus in the urine.[2]

Hyperparathyroidism is hereditary. More women develop hyperparathyroidism than men, especially women who have gone through menopause. Hyperparathyroidism is also more common in older people. People who have vitamin D deficiency are also at an increased risk of developing hyperparathyroidism, because vitamin D helps body absorb the calcium in bloodstream. People who take lithium (typically prescribed to treat bipolar disorder) are also at an increased risk for hyperparathyroidism.[3]

The main symptom that people with hyperparathyroidism complain about is fatigue. The vast majority of patients have some psychosomatic symptoms such as lack of energy, sadness, inability to sleep well, anxiety, nervousness, irritability, etc that their illness seems more consistent with the diagnosis of depression. High blood pressure is another indication of hyperparathyroidism. Nearly 75% of patients with parathyroid disease will have hypertension as a sign.[4]

The effects of hyperparathyroidism on bone are numerous. Excess PTH results in an increase in bone breakdown by means of osteoclastic resorption with subsequent fibrous replacement and reactive osteoblastic activity. The bone may have micro fractures, with subsequent hemorrhage and growth of fibrous tissue and an influx of macrophages. The resulting mass is called gaint cell tumor or more popularly a brown tumor because of the brown color of the vascular elements and blood in the mass. The process of bone resorption and fibrous replacement results in the characteristic radio logic features of generalized bone demineralization, resorption, cysts, brown tumors, erosion of the dental lamina dura, and pathologic fractures.[3]

In this series just in one case the parathyroid lesion became observable due to significant increase in the size of parathyroid gland otherwise they are rarely obvious. It was a fatal malignant lesion. This is considered to be a rare tumor. [5] It was difficult to diagnose in part because of its rarity, lack of definitive diagnostic markers and overlap-ping clinical features of benign primary hyperparathyroid-ism. In great number of cases which lead to the suspicion of hyperparathyroid state were the bony tumorous masses involving mandible, maxilla, and tibia and femur .In one case reported earlier there was a concurrent involvement of maxilla and tibia with a parathyroid adenoma.[6] Giant cell tumor of the bone is a rare primary bone tumor that affects young adults. End of long bones, particularly distal femur and the proximal tibia are preferred locations. Though pathologically benign, these tumors clinically behave aggressively. Hyperparathyroidism promotes giant cell tumor (GCT) formation and its control allows the bone changes to revert. In the facial skeleton GCT are frequent in mandible while the maxilla is exceptionally rare.[7]

There is a well established relationship between primary hyperparathyroidism and recurrent calcium-containing calculi. Traditionally, the diagnosis is confirmed by the presence of elevated parathyroid hormone (PTH) and serum ionized calcium levels. In this series this was established in only one case. In a study published from Pakistan on hyperparathyroidism it was observed that thirty-five percent of patients had renal stone disease, 32.4% had bone disease alone and 27% had both bone abnormality and stones. There were neither bone disease nor stones in 5.4% of patients.[8] Serum Calcium level were close to normal points in most of the cases in this group of patients. This can be explained possibly as to be due to a transient dissociation between the bone resorption and formation with the raised circulating PTH.Patients with primary hyperparathyroidism may have a decreased suppressibility of PTH secretion. One study showed normal or hypocalcaemic state in primary HPTS questioning the credibility of serum calcium as a screening tool.[9]

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Ultrasound, MIBI and Tetrofosmin scans were employed to confirm the presence of parathyroid pathologies. Study indicates that the MIBI scintigraphy is a very sensitive tool for pre-operative localization of hyperplasic parathyroid gland and should be used as the first imaging method. The association of MIBI and ultrasound is recommended because it increases the sensitivity for preoperative hyperplasic parathyroid glands identification.[10] Although adenomas less than 1 cm may be difficult to visualize sonographically, knowledge of typical imaging characteristics of parathyroid adenomas and use of special sonographic techniques will facilitate identification in most patients.[11] Comparison between MIBI and tetrofosmin showed tetrofosmin has the same success rate as sestami-bi for detection of parathyroid adenomas.[12] Though standard text suggest that the overall sensitivities of both radiopharmaceuticals are similar, yet some recent work questions the accuracy of Tc-99m tetrofosmin to detect abnormal parathyroid glands.[13]

It is appreciable that 6 of the seven cases in this series had uneventful parathyroid surgeries. Postoperative health-related quality of life improved significantly. Among patients with preoperative symptoms of depression and anxiety, both symptoms were alleviated significantly at the 12-month follow-up. Surgery for PHPT has been reported to be effective in reducing neuropsychological morbidity associated with PHPT.14 Parathyroid surgery, the definitive treatment for PHPT, has been shown to increase bone mineral density and appears to reduce fracture risk and recurrence of secondary bone tumours.[15] In the current group of patients there was no recurrence reported in the 2 year follow-up period, the result quite in conformity with the recent published material.[16]

One issue which bothers mind with reference to the cases in this small series is the failure to pick parathyroid lesions earlier.It was the presence of sizable co-morbids which mandated a search for parathyroid lesions.Awareness needs to be generated on the possibility.Every suspicious bony pathology and unexplained renal calcinosis should always have a serum parathyroid hormone levels checked.

The other issue being the fact that skeletal manifestations of hyperparathyroid disease should regress spontaneously once the parathyroid adenoma has been resected. Why should one excise the bony co-morbids. Olgard and Lewin reviewed the issue and opined that bone histology can be improved or normalized after treatment that diminishes PTH levels.[17] A significant reduction in the maxillary manifestation has been reported following removal of a parathyroid lesion.[18] How can one wait for a spontaneous regression if the bony lesion is grossly disfiguring or functionally symptomatic? Time of regression cannot be predicted. This justifies the concurrent removal of massive maxillary,mandibular and functionally compromising tibial lesions in this series.The issue of patient satisfaction is also pertinent when the patient desires a quick resolution and cannot wait for natural regression.This point of view has also been advocated in the published material.[19]

CONCLUSION

This small study is very clearly reflective of the fact that PHPT should be suspected in bony tumorous masses and recurrent urolithiasis. For all unexplained bony lesions parathyroid lesion screening is mandatory. The pathology is parathyroid adenoma with secretion of excessive parathyroid hormones. Ultrasound scan screening with t99 Sestamibi scanning are most helpful in establishing the diagnosis. Parathyroid surgery is safe in experienced hands and is rewarding for reverting the morbidity associated with pathologies. If the bony co-morbids are disfiguring and compromising the function it should be surgically excised, particularly if the patient cannot wait for a natural regression. after parathyroid surgery.

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ORIGINAL ARTICLE

SONOGRAPHIC MEASUREMENT OF NORMAL RENAL SIZE AND CORRELATION WITH SOMATIC VARIABLES IN SUBSET OF KARACHI PEDIATRIC POPULATION

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ABSTRACT

Background: Multiple renal pathogenesis manifested as unilateral or bilateral size changes, therefore there should be normative reference data for proper comparison. Our goal is to determine normal standard value ranges for renal dimensions in pediatric population in Karachi. Sonographically renal length in 150 children were measured and correlate with age, gender, body height, weight and BMI.

Objective: Sonographic Measurement of Normal Renal Size and Correlation with Somatic Variables in Subset of Karachi Pediatric Population.

Methods: A six month cross sectional hospital based assessment of kidney size (length, width) was evaluated with the help of sonography. XarioTM 200 Toshiba with convex 3.5 frequency transducer will be used. The mean renal dimensions with standard deviation (SD) were estimated for every group of age. The renal length and width were determined and corresponded with different somatic variables. Descriptive statistics with Regression analysis was done.

Results: The normal length and the width of kidneys and its ranges were obtained. Right kidney length moderately and significantly correlated with height and weight (r=0.651, r=0.654) and age (r=0.538) respectively. However, moderately insignificant with BMI (0.129). Lefts kidney moderately and significantly correlated with height and weight (r=0.665 r=0.705), negative insignificant with age (0.564) and moderately weak insignificant relationship with BMI (0.174).

Conclusion: The research presents the normal range parameters of renal size and measurements by sonography in healthy pediatric population in Karachi. Guideline measurements of kidneys represent a statistically important and comprehensive interaction with specifications of growth which allows us to easily calculate the renal size by derived regression analysis.

KEY WORDS: Renal Size, Sonography, Children, Chronic Renal Disease.

INTRODUCTION

Renal size and function determined the health status of the kidney[1]. Proper body developments and functions are directly related to organ growth rate. The growth rate of renal length will be evaluated with help of distinct measurements like weight, height and anthropometric parameters such as body mass index (BMI).[2] It can be helpful and facilitate us for follow up for the treatment of children with chronic pyelonephritis, obstructive uropathy , and chronic glomerulonephritis in early childhood.[3,4] Sonography helps in accessing and following the patients of urolithiasis, cystic kidney

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