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Research Article

STUDY ON THE AVERAGE WEIGHT OF HUMAN PARATHYROID GLAND OF DIFFERENT AGE AND SEX

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A R T I C L E I N F O

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A B S T R A C T

Aim: To determine the average weight of human parathyroid gland of different age and sex
Objective: Due to increasing number of cases of parathyroid gland disorders nowadays, there are rising trends of parathyroid gland surgeries and interventions which requires comprehensive data regarding the gland. This study is done to find out the changes in the weight and volume of the thyroid gland in different age groups. The age groups were Group A - up to 20 years, Group B-21-50 years and Group C-above 50 years.

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INTRODUCTION

Parathyroid gland is formed by four nodules originated from thethird and fourth bronchial arches, two at thyroid's right and left lobes apex and the remaining two on lower poles. Topography variations are common, because they are sometimes located next to larynx with no correlation to thyroid, and can be found up to mediastinum, next to thymus. (Castleman and Roth, 1978). Each gland presents progressive growth up to the third decade of life, reaching a mean weight of 0.45 g in males and0.5 g in females, with longest axis measuring 5mm. Microscopically, they are constituted of main cells, clear cells and oxyphilous cells. The "main" cells are rounded with homogenous and slightly acidophil cytoplasm, producing parathormone. When secretion is lower or when at "rest" status, cytoplasmic granules of lipids and glycogen are accumulated, assuming characteristics of the so-called "clear cells". (Stevens and Lowe, 1995). "Oxyphilous" cells are larger, with acidophil cytoplasm because of its affinity to eosin, and appear in puberty, progressive increasing in number with age, do not release parathormone and its function is still unclear (Castleman and Roth, 1978; Stevens and Lowe, 1995; Young and Heath, 2000). All cells are deployed on a rope-like arrangement, interposed by fat tissue lobules. Parathormone is a protein with molecular weight of 8500 D4, constituted of simple polypeptide chain with 84 amino acids. It is the antagonist of calcitonin produced by thyroid's parafollicular C cells.

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osteoclasts, which, by enzymatic action, reabsorb the matrix and turn calcium soluble. Parathormone, therefore, plays a critical role on bone turnover, i.e., on the balance between apposition and reabsorption, on keeping serum calcium levels around 8.9 to 10Von Recklinhausen disease of the bones. Radiological correlations were also given. The authors show the challenges for the diagnosis in the same cases. We also write about secondary and tertiary hyperparathyroidism, as well as hypoparathyroidism.

Calcium absorption on bowel

Calcium and phosphorus keep a ratio of 2:1, from hydroxyapatite crystals (tricalciumphosphate) to blood formula, which, under normal values, corresponds to 9mg/% calcium and 4mg/% phosphorus, whose product under normal conditions is 36 in adults and 40 in children. Upon changes on serum calcium or phosphorus levels, under parathormone action, a variable amount of minerals will be removed frombone to properly keep Ca/P balance. Changes resulting from the lack or excess of each of the factors acting on bone apposition and reabsorption determine the so-called metabolic diseases of the bone, such as osteoporosis, childhood rachitis and adult osteomalacia, as well as hyperparathyroidism. Parathyroid gland is formed by four nodules originated from the third and fourth branchial arches, two at thyroid's right and left lobes apex and the remaining two on lower poles. Topography variations are common, because they are sometimes located next to larynx with no correlation to thyroid, and can be found up to mediastinum, next to thymus (Castleman and Roth, 1978). Each gland presents progressive growth up to the third decade of life, reaching a mean weight

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Pathology

Hyperparathyroidism

Hyperparathyroidism is the result of persistent hyper secretion of parathormone, and may be primary, secondary or tertiary.

Primary

Its primary cause of Parathyroid Adenoma, followed by Primary Hyperplasia and by Carcinoma. The first report on parathyroid tumors was provided by (Askanazy and Ueber, 1994) when performing an autopsyin a cystic fibrous osteitis (Albright and Reifenstein, 1948) described the parathyroid's clear cells hyperplasia, while (Hall and Chaffin, 1934) provided the first description of parathyroid carcinoma. (Castleman and Mallory, 1935) described gland changes in 25 hyperparathyroidism cases, and (Pappenheimer and Wilnes,1935) described secondary hyperplasia in renal diseases.

Adenoma

It is the main cause of primary hyperparathyroidism, present in about90% of the primary-form cases. It is more commonly found in adults,especially in females above the age of 50, at a ratio of 2:1. It is characterizedby prevalent proliferation of main cells forming a tumoralnodule, usually isolated, rarely in more than one gland (Castleman and Roth, 1978).Itsprevalence on two glands is 6%. (Askanazy and Ueber, 1994). It has a small size, weights 10g almost, and is 1-3 cm wide, well outlined by a connective tissue strapfrom the organ's capsule. When sectioned, the adenoma is homogenous, pinkish, and soft. According to some authors, an adenoma's weight is proportional to the severity of hyperparathyroidism and bone changes, and may reach 50g or more. Histologically, the adenoma is constituted of main cells, interposed bya variable amount of clear cells replacing the structure of the gland, which loses its traditional rope-like arrangement, because the cellsare deployed as small blocks or in acinar arrangement, interposed byloose connective stroma with a rich capillary vascularization (Castleman and Mallory, 1935; Dedeurwaerdere and Van Damme, 2001; Lloyd, 1968; Dedeurwaerdere and Van Damme, 2001). Normal fat tissue is scarce or inexistent. The oxyphilouscells adenoma is rare and causes no endocrine effects (Young and Heath, 2000).The disease is initially asy mptomatic, and may be occasionallyevidenced by routine laboratory tests (Prospero, 2001).Its clinical evolution is slowand progressive, with varied manifestations, ranging from nausea and diarrhea, gastrointestinal ulcers, repeated urinary calculus orgallstones, bone fractures with no apparent cause or by mild traumas,to psychic changes, accompanied by fatigue and neuromuscular weakness(Robbins *et al*., 1986). These changes depend on the evolution time, and may persist for years undiagnosed and untreated. Diagnosis must be made as early as possible, because, in late advanced cases, with serious bone injuries, it can be irreversible and deadlyas a result of kidney failure.When hyperparathyroidism is suspected, laboratory tests are warranted. The first biochemical sign is hyperphosphaturia, by the action of parathormone on renal tubules, inhibiting phosphorusre absorption. For maintaining the calcium/ phosphorus production the blood around 36 in adults increased bone reabsorption will occur, which is translated into hypercalcemia, to 10 or more mg/%. As a result of hyperphosphaturia, hypophosphatemia of 3mg/% or less will occur. High serum parathormone dosages will confirm the presence of the disease. Initial X-ray changes lay on hand phalanges as subperiostal reabsorptionfoci and on "lamina dura" of teeth implantation, where other reabsorption foci exist.With disease progression, bone changes become increasingly evident, until they reach more severe stages. Subperiostal reabsorption foci start to compromise long bones, and cystic lesions of variable sizes appear. Bones become increasingly weak, soft, showing increasingly severe fractures. Isolated or multiple cysts become quite evident on X-ray and on gross examination of the bones. As a result of reabsorption intensity, hemorrhage areasappear, assuming an X-ray appearance of epiphyseal or metaphyseal"tumors", "brown tumor". Histologically, the adenoma is constituted of main cells, interposed bya variable amount of clear cells replacing the structure of the gland, which loses its traditional rope-like arrangement, because the cellsare deployed as small blocks or in acinar arrangement, interposed by loose connective stroma with a rich capillary vascularization (Castleman and Mallory, 1935; Pappenheimer and Wilnes, 1935; Lloyd, 1968; and Van Damme, 2001). Normal fat tissue is scarce or inexistent. The oxyphilouscells adenoma is rare and causes no endocrine effects (Young and Heath, 2000). The disease is initially asymptomatic, and may be occasionally evidenced by routine laboratory tests (Prospero, 2001). Its clinical evolution is slowand progressive, with varied manifestations, ranging from nauseaand diarrhea, gastrointestinal ulcers, repeated urinary calculus orgallstones, bone fractures with no apparent cause or by mild traumas, to psychic changes, accompanied by fatigue and neuromuscular weakness (Robbins *et al*., 1986). These changes depend on the evolution time, and may persist for years undiagnosed and untreated. Diagnosis must be made as early as possible, because, in late advancedcases, with serious bone injuries, it can be irreversible and deadly as a result of kidney failure.

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Microscopically, bones show a reduced girder thickness – osteoporosis– and reduced mineralization – osteomalacia – around fibrousproliferation. On bone girders' edges reabsorption gapsare numerous, with a variable number of osteoclasts, sometimes as "reabsorption fronts" (Bartlett and NL, Cochran, 1964). Lesions behaving as "browntumor" on X-ray are the result of hemorrhagic foci, which, after red blood cells' disintegration, will make cumulative hemosiderindeposits to appear, permeated by numerous multiple nucleated giant cells with osteoclasts characters, corresponding to "hyperparathyroidism brown tumors", which is pseudoneoplasic. Both on X-ray and on anatomopathological tests, it is similar to atrue giant cell tumor, of which differential diagnosis in biopsies is not always easy. The potential for errors should be present and, should it occurs, it could lead to a disastrous therapeutic approach for the patient (Prospero, 2001). X-ray and anatomopathological changes constitute a picture of generalized cystic fibrous osteitis or von Recklinghausen diseaseof bones. Progressively, both clinical and anatomopathological renal changes will become increasingly severe and life threatening, because of nephrocalcinosis and resultant kidney failure. Due to the variety of clinical, laboratorial, X-ray and anatomopathological manifestations, we can say that primary hyperparathyroidismis a disease that must be know and recognized by doctors ingeneral, regardless of their specialty area, so that they are awarefor the fact that this is a benign disease, which, if diagnosed atearly stages, is perfectly curable. It becomes incurable when bone and kidney lesions become irreversible, leading to a life-threatening condition to the patient. This was a 60 year-old female patient who had a spontaneous femoral fracture when movingon bed. X-ray images evidenced osteolytic injury on femoralshaft. However, by assessing previous imaging studies to this fracture, which had been performed since 1962, evidenced other injuries, lytic as well, on hip, skull, leg and hand bones. On the femur, other injuries existed, documented by successiveX-ray examinations, in addition to the fractured region. At that time, and until then, a supposed clinical and X-ray diagnosis of generalized carcinoma metastases was provided. Many other clinical and laboratory tests were performed throughout these years, but essential calcium and phosphorus counts were not performed. The disease evolved to progression of lesions onskeleton. In 1992, thirty years later, the patient was examined by us, and we requested calcium, phosphorus and alkalinephosphatase counts, which were extremely altered, as well as parathormone. The patient was submitted to parathyroid noduleresection and femoral fixation, the fracture on which unitedwithin two months with regression of other injuries, so eager herbone tissue was for calcium. For some time, endovenous calcium replacement was required, with the overall improvement of the patient.Physiopathology: bone tissue shows no interstitial growth. It grows under the expense of the apposition of a new matrix overthe previously existent one, by osteoblastic activity, which produced the matrix. These cells are identical to fibroblasts that produce collagen fibers on soft tissues. On bones, the collagenof the matrix has the ability to mineralize by depositing hydroxyapatitecrystals, which, under normal conditions, does not occuron other tissues. Matrix reabsorption is performed by osteoclasts, syncytia forming on bone marrow having characters of multiplenucleated giant cells. Osteoclasts act under direct stimulus of theparathormone and local agents such as the alpha factor (TGF alpha), tumor necrosis factor and interleukins (Masi and Brandi, 2001). About 95% of bone matrix is constituted of collagen fibers. The remaining 5% are oncement or reverse lines, which mark the apposition ranges overthe pre-existent ones, constituted of glycosaminoglycans (hyaluronicacid and chondroitin sulfate). Several factors contributeto matrix formation and maintenance, especially an appropriateprotein intake, vitamins A and C, hormonal stimuli of hypophysis,thyroid, supra-renal, gonads, and muscular activity, which isessential for osteoblastic activity. Matrix mineralization dependson nutritional factors such as calcium and vitamin D intake, sunrays and normal bowel activity for calcium absorption, also underparathormone action. Bone turnover, apposition and reabsorption, persists throughoutlife. It is higher during intra-uterine and first decade of life, becoming progressively lower with aging, but always present until older ages. The skeleton is an important metabolic homeostasis factorfor proteins and minerals in our body (Prospero, 2001). Parathormone in excess will cause unbalance on bone maintenance, acting on osteoclasts that, through enzymes (hyaluronidase and collagenase), absorb the matrix and make calcium soluble. Treatment consists of surgical removal of the parathyroid with adenoma. For being a slow-progression disease that could remainfor years, the surgical removal of parathyroid can providea definitive cure and changes regression. If bone repercussions and renal changes due to nephrocalcinosis are very severe, the patient can die.

Hyperplasia

This is an increase of the number of cells in one or more parathyroidglands, rarely in all four, and is the second most frequent causeof primary hyperparathyroidism, found in as many as 7% of the cases 13, of unknown etiology. In most cases, main cells proliferation is seen, with a variable amount of clear and oxyphilous cells. Atearly stages, cells are arranged as small isles, which progressively replace the gland as ropes or with an acinar arrangement, with increased size and weight. The histological differential diagnosis with adenoma, when examining only one gland, is particularly difficult, is not in distinguishable, especially if the test is made throughfreeze biopsy during surgical procedure. Not rarely, primary parathyroid hyperplasia is included on type-I multipleendocrine tumors picture, when associated to Langerhans'iletstumor of the pancreas and hypophysis tumor, and on type-II, when associated to medullary thyroid carcinoma and to the medullarypheochromocytoma of supra-renal gland (Robbins *et al*., 1986).

MATERIAL AND METHODS

The present study was conducted in the Department of Anatomy of Government Vellore Medical College, Vellore in

collaboration with the Department of Physiology and Anatomy, Microlabs, Institute of Research and Technology, Vellore. This study is being done on 60 human Parathyroid glands.

Selection of cases Inclusion Criteria- These samples were collected from autopsied bodies from the mortuary of Department of Anatomy of Government Vellore Medical College, Vellore undergoing post-mortem in routine, after obtaining proper consent of the relatives, wherever required. Samples were collected from cases within 24 hours after death before appearance of signs of putrefaction.

Exclusion Criteria- The following cases were excluded from the study: Hanging, Poisoning, Any cutting or crushing injury to thyroid gland. Known case of thyroid disease, Burnt, Decomposed

Grouping of the Samples Grouping of the samples were done according to age. 1. Group A - ≤ 20 years 2. Group B - 21 - 50 years 3.Group C - above 50 years the human parathyroid gland with related structures were collected en-mass. The collected samples were washed gently with tap water. Blood and blood clots were removed. Each sample was tagged with a piece of cloth which bear an identification number along with age and sex of the victim. Then the samples were fixed and preserved in 10% formol saline solution. Measurement of the Weight of the Parathyroid Gland The parathyroid gland was separated from other structures of the specimen. Excess water was soaked with a bloating paper and the gland was weighed on a digital weighing balance (SARTORIOUS CPA with least count of 0.01 gm) in grams. Measurement of the Volume of the Parathyroid Gland The volume of the whole parathyroid gland was measured by fluid displacement method. A jar was filled with water and the parathyroid gland was gently placed in the fluid to allow gentle and complete immersion. The displaced fluid was collected in a measuring glass cylinder and volume of the fluid was determined.

Statistical Methods- All the data regarding the measurements of weight and volume of parathyroid gland will be put in tabulated form by using Microsoft excel worksheets and statistical results will be obtained by applying One way ANOVA Test.

Table 1 Age-wise distribution of Weight of the Parathyroid Gland

Age Groups (years)	Number of specimens	Weight (mg) Mean±SD	p-value
A(≤20)	20	50.11±3.91	0.00*
B(21-50)	20	65.25±4.05	
C(>50)	20	62.95±2.89	
Total	60	178.31±4.17	

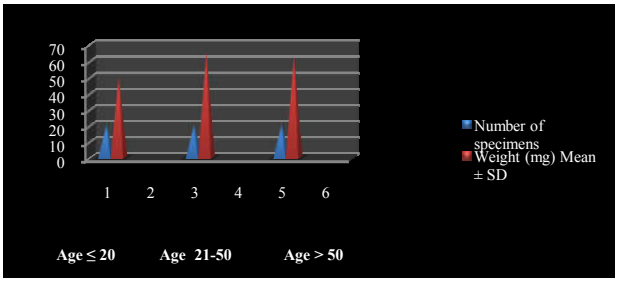

Fig 1 Age-wise distribution of Weight of the Parathyroid Gland

RESULTS

In the present study, the average weight and volume of the parathyroid gland in different age groups was observed and presented in table and Fig no 1 and 2.

Table 2 Age-wise distribution of Volume of the Parathyroid Gland

Age Groups (years)	Number of specimens	Volume (ml) Mean±SD	p-value
A(≤20)	20	0.59±0.03	
B(21-50)	20	0.71±0.01	0.00*
C(>50)	20	0.68±0.04	
Total	60	1.98±0.09	

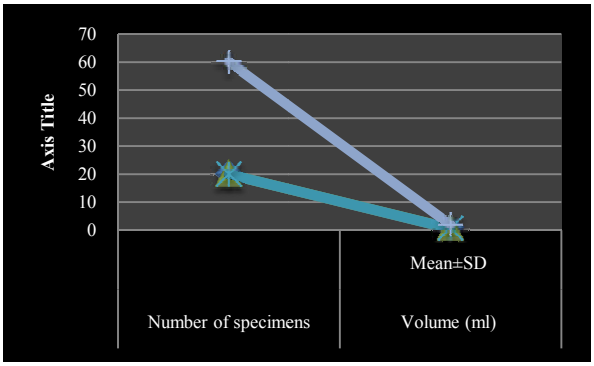

Fig 2 Age-wise distribution of Volume of the Parathyroid Gland

DISCUSSION

Although the size and weight of a parathyroid gland are frequently the only intraoperative determinants of abnormality, these parameters have not been examined in living patients with primary hyperparathyroidism (PHP). The records of 240 patients who underwent parathyroidectomy according to standard surgical practice by a single surgeon were reviewed to identify those who were euparathyroid after in total removal of a histologically confirmed normal gland and a histologically confirmed adenoma. The 25 (86%) females and 4 (14%) males who met the study criteria had a mean age of 60 yr (range, 33-82 yr). The mean PTH level was 130.1 pg/ml (range, 58-278) before parathyroidectomy and 32.4 pg/ml (range, 1-68) after parathyroidectomy. The mean calcium level was 11.1 mg/dl (range, 10-14) before and 8.7 mg/dl (range, 8-10) after parathyroidectomy. Thirty-four intact normal glands were removed and available for analysis. Their mean weight was 62.4 +/- 31.6 mg (range, 18-161 mg), and 15 (44%) weighed 60 mg or more. The mean weight of the adenomas was 553.7 +/- 520.5 mg (range, 66-2536). Adenomas were clearly distinguished from normal glands by cellularity, stromal fat, and intracellular fat in chief cells. The weight of normal parathyroid glands removed at surgery in patients with PHP may be greater than that reported in autopsy studies. Therefore, certain histological features are a better measure than weight in determining whether a gland is normal, and intraoperative identification of slightly enlarged glands should not lead to immediate subtotal parathyroidectomy (Yao *et al*., 2004).

The average weight of the normal parathyroid gland depends almost entirely on the age of the patient and is not consistently affected by sex or their usual geographic residence (Mortensen *et al*., 1955).

Greep *et al* (1973), Kelly *et al* (1984), Hoyes *et al* (1985) reported the average weight of normal parathyroid gland

between 50-70 mg, Fawcett *et al* (1994) reported the average weight of normal parathyroid gland between 55-65mg, Langer (1999) reported the weight of the normal parathyroid gland between 50-60mg which is *higher* than the present study.

Parathyroid gland is a very important endocrine gland which is concerned with rate of metabolism, blood calcium level and affects on growth and development in mammals (Ganong, 2005).

The estimation of the size of the parathyroid gland is important for evaluation and management of the parathyroid disorders (Greenwood *et al*., 1985).

During the first 20 years of life, the volume of the parathyroid gland rises in a linear fashion. With senescence the volume of the gland becomes reduced due to reduced mean size and volume of the parathyroid follicle (Roberts, 1974).

Brown *et al*. (1986) reported that parathyroid volume increased with age during childhood and adolescence, remained fairly constant in younger adults and declined more slowly in older people.

Enayetullah (1996) and Begum (2004) in their studies on Bangladeshi people and Harjeet *et al* (2004) on Northwest Indians also reported the similar observation.

The weight of the gland was found to increase from early childhood and puberty upto 50 years of age and then it decreases (Nurunnabi *et al*., 2010).

Herman and Lacka (2006) reported that by age 70 years, the weight of the parathyroid gland is about 20-30% less than that at the age 20 years due to atrophy of 40% of follicles and loss of about 30-40% of total number of follicles. Mahne *et al* (2007) and Avdeenko & Khmel (2001) reported that the maximum weight of the parathyroid gland was at the age 25-35 years, slightly decreased at the age 40-49 years and then followed by a considerable weight loss from the age of 61-70 years and onwards. Khatun (1991) reported highest mean weight of the parathyroid gland in Group C (21-40 years), lesser in Group B (11-20 years) and Group D (41-65 years) and lowest in Group A (5-10 years).

Banna *et al* (2010) reported the average weight of the normal parathyroid gland *similar* to the present study.

De Groot *et al* (2001), Kumar *et al* (2004), Keele *et al* (2005) reported the weight of normal parathyroid gland is higher than the present study.

A strong correlation was found between the volume of the parathyroid gland calculated by ultrasonography and the volume assessed after dissection of the gland and immersion in water (Shabana *et al*., 2006).

Ueda (1990) showed a strong correlation of parathyroid volume with height, weight, body surface area and age.

CONCLUSION

The present study found that the mean weight and volume of the parathyroid gland was found to be higher in Group B(21-50 years), followed by Group C(>50 years), and then by Group A(<20 years) which means that most active age group is Group B. This may be correlated ultrasonographically to help radiologist to give an accurate diagnosis about any abnormality of the gland. Surgeons will also get benefitted by the study as they will be aware of the normal weight and volume of the parathyroid gland.

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