MALIGNANT TUMOURS OF THE THYROID GLAND

Essay

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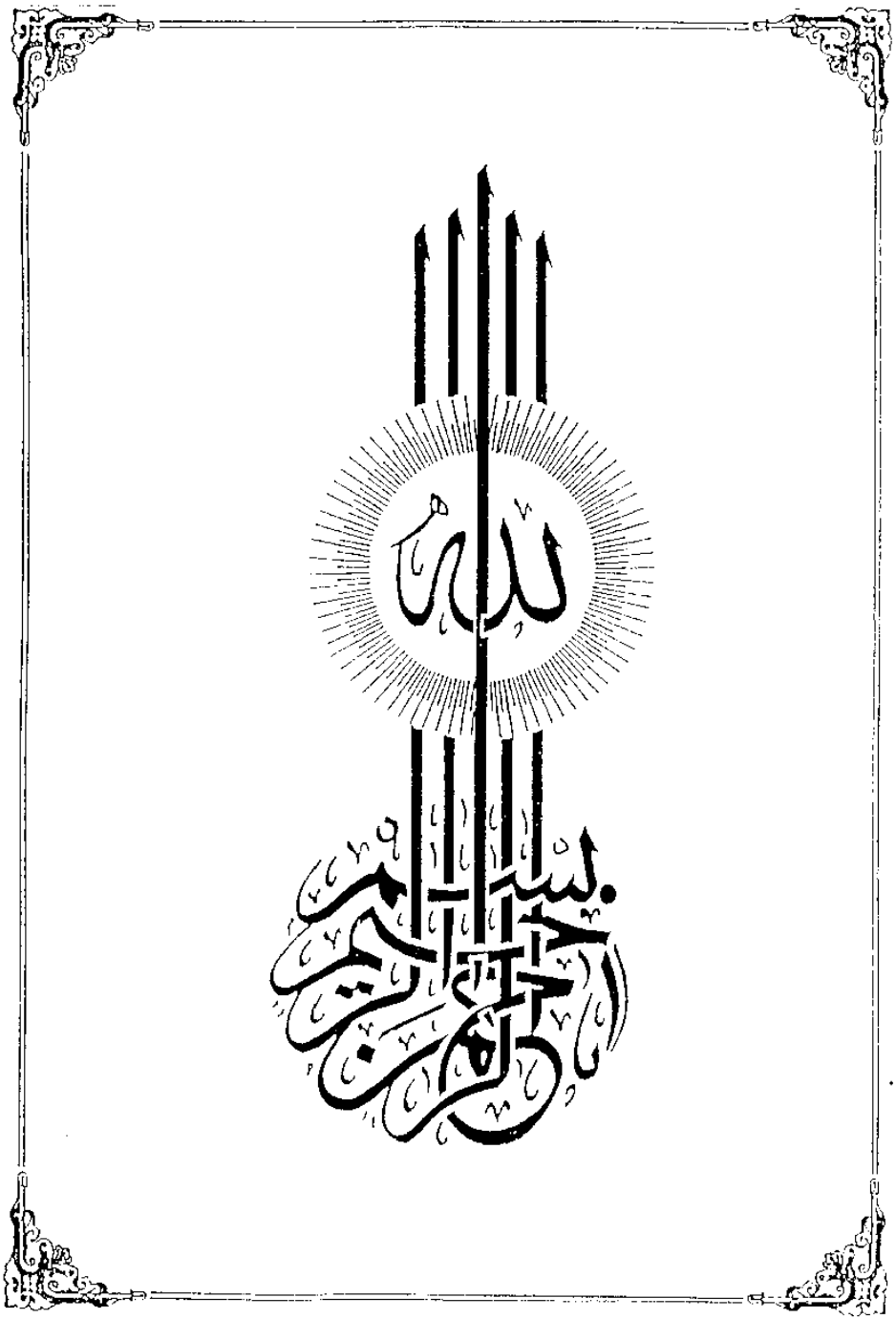

CONTENTS

INTRODUCTION

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Malignant tumours of the thyroid gland its importance lies in the fact that it behaves in an unusual manner both as regards its wide histological variations and its clinical evaluation. For this reason it has aroused our interest which comes to be out of proportion to its low indicence.

From the geographical point of view it seems that there is a marked difference in the incidence and behavior of thyroid carcinoma. The work presented in the Essay is an attempt to add a little to our understanding and knowledge of the problem as goiters is an endemic disease in Egypt.

Currently, malignancies are present in 12 to 14 % of patients undergoing thyroid surgery because of suspicion of cancer. Similarly, carcinoma is seen more frequently in a thyroid gland that is considered clinically to have a single nodule than to be multinodular.

From the pathological point of view, carcinoma of the thyroid gland shows an extremely variable rate of growth unequaled by any other type of cancer. Thus, the spread of the highly differentiated papillary carcinoma of the thyroid in the cervical lymph glands may be so

slow as to last many years. On the other hand a highly anaplastic carcinoma spreads very rapidly and may cause death within few months. This variation raises special problems as regards the line or lines of treatment. Thus, the proposal that all goiters and all nodules should be subject to surgery probably is justifiable.

Because of all these difficulties, these work has been carried out in an attempt to review the literature concerning the problem of malignancy in the thyroid gland.

REVIEW OF LITERATURE

ANATOMY OF THYROIDGLAND

* Development:

The thyroid gland first appears in early somite embryos as a thickening of the endoderm of the floor of the pharynx. This thickening forms a diverticulum attached to the buccal cavity by the thyroglossal duct. During the seventh week, the thyroid reaches its definitive location anterior to the first six tracheal cartilages. Whether or not part of the thyroid gland is formed by epithelial proliferation of the fourth pharyngeal pouch is still a point of discussion. (Timothy's et al., 1979). However, It is believed that the parafollicular cells, which produce calcitonin may develop from the fourth and even the fifth pharyngeal pouch.

* Gross anatomy of the thyroid gland:

The gland consists of two symmetrical lobes united in front of the second, third and fourth tracheal rings by an isthmus. Each lobe is pear shaped, consisting of a narrwer upper pole and a broader lower pole that lies under cover of the sterno thyroid and sterno-hyoid muscles. The upper pole lies tucked away beneath the upper end of the sterno-thyroid muscle, between it and the ala of the thyroid cartilage. The lower pole extends along the side of the trachea as low as the sixth tracheal ring.

The gland is invested in a delicate histological capsule, and lies within an envelope of the pretracheal fascia. A small portion of the gland often projects upwards from the isthmus and to the left of the midline named the pyramidal lobe. It is attached to the inferior border of the hyoid bone by fibrous tissue muscle fibres, the levator glanulae thyroideae. Sometimes seperate masses of thyroid tissue are not uncommonly found near the hyoid bone, in the superior media-stinum, or beneath the sterno-mastoid muscle.

* Microscopic anatomy:

The follicles are the units of structure of the thyroid gland, which are lined by a single layer of epithelium. The shape of these cells varies from flat in a resting state and are tall when the gland is active. The follicles contain colloid which consists of a glycoprotein called thyroglobulin. Thyroglobulin is synthesized by the ordinary cells of follicles and secreted into the lumina of follicles. In between the follicles lie small round cells, the parafollicular or c cells which secrete calcitonin. (Cormack et al., 1979).

* Blood supply:

(1) Arteries:

The main arterial blood supply is derived from the

superior and inferior thyroid arteries. The superior thyroid artery the first branch from the anterior aspect of the external carotid artery, after giving off its sterno-mastoid and superior laryngeal branches, pierces the pre-tracheal fascia to reach the upper pole of the gland where it divides into anterior and posterior branches. The anterior branch runs down to the isthmus and the posterior branch that runs down the back of the lobe and anastomoses with an ascending branch of the inferior thyroid artery. The inferior thyroid artery is a branch of the thyro-cervical trunk, divides outside the pretracheal fascia into four or five branches that pierce the fascia to reach the lower pole of the gland. The recurrent laryngeal nerve lies normally behind these branches, but it is common for it to pass between them before they pierce the pretracheal fascia. The thyroidae ima artery enters the lower part of the isthmus in 3 % of individuals. It springs from the brachio-cephalic trunk or direct from the arch of the aorta.

(2) Veins:

The venous return of the thyroid gland occurs via the following veins:

i- The superior thyroid vein from the upper pole and it enters either the internal jugular or common facial vein.

ii- The middle thyroid vein passes from the middle of the lobe directly into the internal jugular vein.

iii- The inferior thyroid veins, from the isthmus and lower poles form a plexus that lies in the pretracheal fascia and drains into the brachio-cephalic veins.

* Lymphatic drainage:

Lymphatic drainage of the thyroid gland follows the arteries. From the upper pole they enter the antero-superior group of deep cervical lymph nodes. From the lower pole they pass with the inferior thyroid artery back to its point of origin into the postero-inferior cervical group. A few may pass downwards into pretracheal nodes.

* Innervation:

The sympathatic supply is derived from the middle cervical ganglion and enters the gland on the inferior thyroid artery ; some fibres from the superior cervical ganglion travel with the superior thyroid artery (Last, 1979).

PHYSIOLOGY OF THE THYROID

The rate of tissue metabolism is controlled by the thyroid hormones which stimulate the oxygen consumption of most cells of the body; regulate lipid and carbohydrate metabolism and they are also necessary for normal growth and maturation.

* Formation and secretion of thyroid hormones:

The principal hormones secreted by the thyroid gland are thyroxine (T_4) and tri-iodothyronine (T_3) which are iodine containing amino acids. Dietary iodine, principally from fish, milk, eggs and iodinated salt or bread, is absorbed from the stomach and upper small intestine as iodide ions. The circulating iodide is trapped and concentrated by the thyroid up to 40 times the plasma level. Within the thyroid cell iodide ions are rapidly oxidised by a peroxidase enzyme to iodine which immediately reacts with tyrosine attached to thyroglobulin in colloid to form mono-iodotyrosine(MIT)and di-iodotyrosine (DIT). thyroxine (tetraiodothyronine, T_4) and tri-iodothyronine (T_3) are formed by coupling of these iodotyrosines.

Hormone containing molecules of thyroglobulin are then stored in colloid till required. Microvilli on the apical surface of the cell engulf colloid droplets (pinocytosis) which fuse with lysosomes whose proteolytic enzymes

hydrolyse thyroglobulin to release thyroid hormones and other iodotyrosines. Most of the latter are split into iodide and tyrosine in the thyroid cell and is mainly thyroid hormone that is released into the blood stream. The majority of circulating thyroid hormone is protein bound but it is the free thyroid hormone, that is metabolically active. thyroxine is more strongly bound than triiodothyronine and this may explain the differences in rapidity of action and turnover. With decreasing affinity, thyroxine binding globulin (TBG), thyroxine binding prealbumin (TBPA) and albumin bind thyroid hormones.

The functions of the binding proteins are regulation of the rate of delivery of free hormones, buffering of changes in secretion or degradation and prevention of loss by way of the liver or kidneys (Jamieson & Kay's 1980).

The thyroid gland secretes more T_4 than T_3 and there is no evidence that T_4 is converted to T_3 in the thyroid. There is, however, good evidence that T_4 is deiodinated to T_3 in other tissues and this is very clearly shown in a thyroitic patients, in whom administration of T_4 leads to the development of normal or raised levels of T_3 in the serum. Stimulation of thyroid activity by injection of TSH or TRH raises the serum levels of both T_3 and T_4. Enhanced thyroidal secretion of T_3 and greater

peripheral conversion of T_4 to T_3 may raise the serum T_3 level 5 - 10 fold. In some thyrotoxic patients the serum T_4 level is normal while that of T_3 is raised (T_3-thyrotoxicosis). From these it seems likely that T_4 appears to act as a stable prohormone for T_3. As T_4 is largely converted to T_3 in the body, this provides a reservoir from which T_3 level can be maintained (Samson wright's, 1984).

* Regulation of thyroid hormone secretion:

To maintain a normal basal metabolic rate, a specific feedback mechanism operates through the hypothalamus and anterior pituitary gland to control the rate of thyroid secretion.

The thyroid-stimulating hormone (Thyrotropin), is an anterior pituitary hormone has the following specific effects on the thyroid gland:-

(1) Increased proteolysis of the thyroglobulin in the follicles.

(2) Increased activity of the iodide pump, which increases the rate of "iodide trapping".

(3) Increased iodination of tyrosine and coupling to form the thyroid hormone.

(4) Increased the size and secretory activity of the thyroid cells.