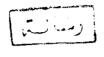
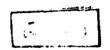
Different Surgical Aspects Of Primary and Tertiary Hyperparathyroidism

Essay Submitted for partial fulfilment of M.S. degree in General Surgery





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وم أونينم من العلم المقلبلي صدق الله العظيم



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Nagy Baliegh Ali



DEDICATION

To...



Nagy Baliegh

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Introduction

Hyperfunction of the parathyroid gland may occur as a primary or a secondary condition. In the primary hyperparathyroidism, it is generally due to an adenoma that may be solitary or a component of multiple endocrine neoplasia syndrome.

(Walter and Israel, 1987)

In the secondary hyperparathyroidism, on the other hand, the hyperplasia of the gland occurs secondary to the hypocalcemia produced, for example, in many types of osteomalacia and rickets or it may occur secondary to a chronic renal disease (Renal osteodystrophy) especially in young people. In the latter condition, The resultant hypophosphatemia, owing to the chronic loss by the diseased kidney, is the stimulus for the excessive Parathormone release and hence the condition is produced.

(Walter and Israel 1987)

Tertiary hyperparathyroidism is the development of hypercalcemia due to adenomatous changes in the parathyroid gland in some cases of long standing parathyroid hyperplasia secondary to malabsorption or chronic renal failure.

(Robbin et al., 1995)

Also, parathyroid hormone may be abnormally high owing to an ectopically - producing focus, the most commonly met with is the squamous cell carcinoma of the lung.

(Walter and Israel, 1987)

Rossi et al., in (1995) stated that, since the introduction of biochemical autoanalyzers, primary hyperparathyroidism is no longer uncommon diagnosis and the diagnosis can be established by clinical evaluation and persistently elevated serum calcium and parathormone levels.

Peck in 1987 reported that, despite the great advance in the imaging techniques for the preoperative localization of parathyroid adenomas, still some surgeons believe that a surgeon's experience is needed in first time exploration of the necks for patients with hyperparathyroidism.

The management of symptomatic primary hyperparathyroidism is straightforward and the disease can be effectively treated with surgery .

(Clark 1988)

Excision of the adenoma, subtotal or total parathyroidectomy with autotransplantation of the parathyroid glands are the current surgical options for treatment of hyperparathyroidism.

(Kaplan, 1994)

Aim of the work:

It is to study the different etiological factors of the hyperfunctioning parathyroid gland with a particular attention to those associated with chronic renal diseases. In addition, we aim at focusing on the most useful localizing tool (or tools) that can be routinely used for preoperative assessment of patients having hyperparathyroidism.

HISTORICAL BACKGROUND

In 1880, a Swedish student, Ivar Sandstrom, First described the Parathyroid glands in several animals and in man and named the Parathyroid glands(glandule parathyroidae). The name was well chosen, as "Para" is the Greek prefix meaning alongside of.

(Petti, 1990).

His discovery went unnoticed until 1891, When the glands were rediscovered by Gley, Who demonstrated that their removal led to tetany. In the same year as Gley's discovery, Von Recklinghausen described a characteristic disease of bone that latter was found to be caused by hyperparathyroid. The first to describe the association of bone disease and parathyroid neoplasia was Askanzy, Who in 1904 studied a women with pain in the extremities and spontaneous fractures. At autopsy, She was found to have both generalized osteitis cytica described early by Von Recklinghausen and an "incidental" tumor lateral to the thyroid glands.

(Wells et al., 1991)

Biologically active Parathyroid extracts were first prepared by Collip in 1925. Parathyroid hormone was isolated and purified by Rasmussen and Craig and Auerbach in 1959. In 1963, Berson and associates developed a radioimmunoassy for parathyroid hormone.

(Wells et al., 1991)

The first parathyroid exploration in the united states was performed in 1926 at the Massachusetts General Hospital on a merchant marine with severe bone disease, Captin Charles Martell. Unfortunately, the abnormal gland was not found until his seventh operation in 1932; and he subsequently died in tetany