SERUM PARATHYROID HORMONE IN PATIENTS UNDERGOING REGULAR HAEMODIALYSIS

T H E S I S

Submitted In Partial Fulfilment For Degree Of Master Of General Medicine

BY

HOSNY ABDEL SALAM GADALLA

(M.B., B.Ch)

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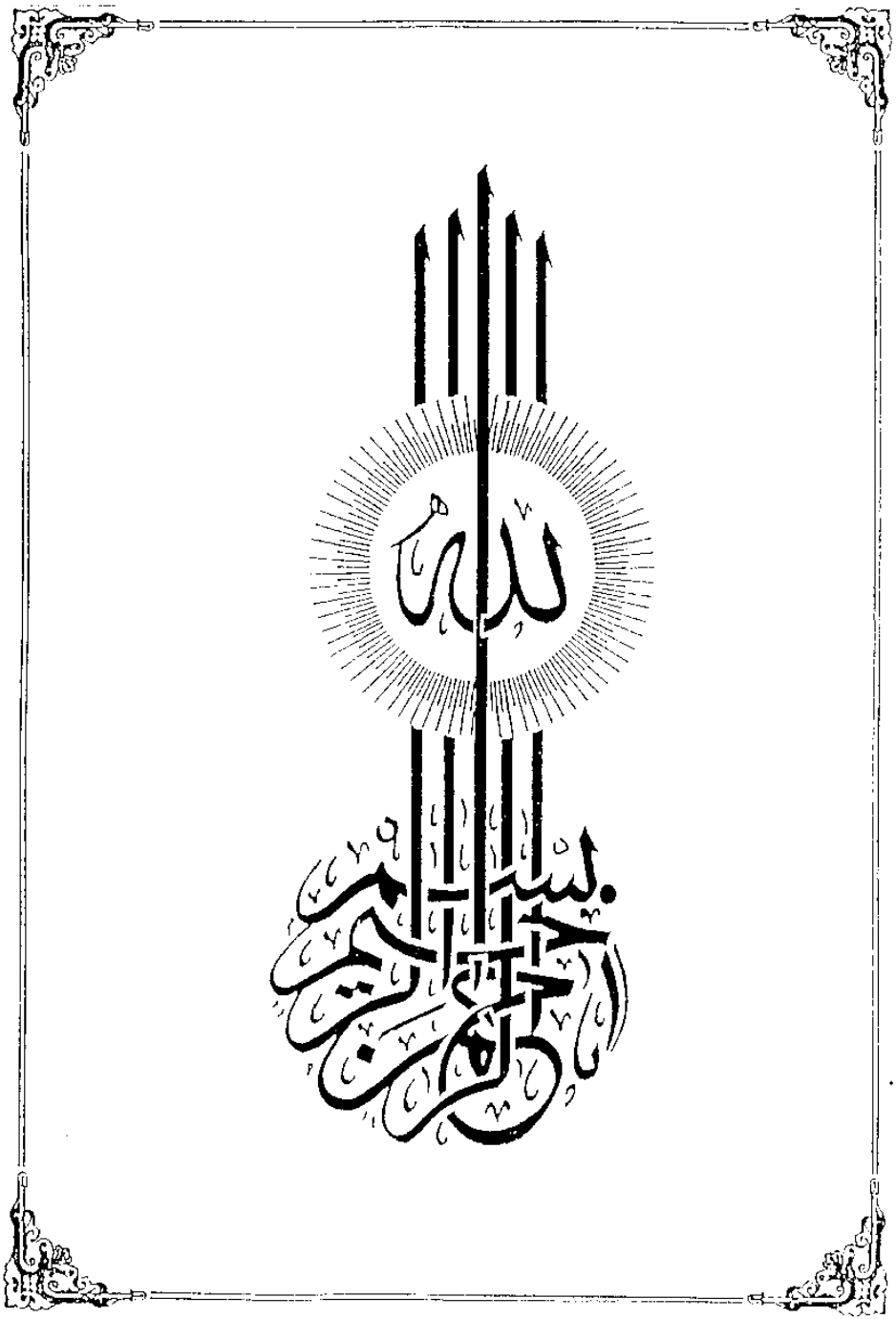

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DEDICATION

THIS THESIS IS DEDICATED TO

MY DEAR MOTHER,

WIFE AND CHILDREN

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INTRODUCTION & AIM OF WORK

INTRODUCTION

The parathyroid gland is well known to play a major role in calcium Homeostosis. Serum parathyroid hormone (PTH) level is often markedly elevated in patients with chronic renal failure (CRF) due to disturbance in calcium Homeostasis leading to negative calcium balance. This view is widely discussed by many authors Coburn, et al., (1969, 1973) & Slatopolsky, et al. (1971, 1972) and Moorhead, et al. (1975). The role of PTH and calcitonin in calcium homeostasis, absorption, turnover, etc. has been discussed by Ghareeb, et al. (1981).

There is a contraversy about the pathogenesis and role of parathyroid gland in bone disease associated with CRF. In Egypt renal osteodystrophy in CRF has been carried out by many investigators, Abdel-Gaber, M. (M.D. Thesis - Assuit University, 1980) and Atalla, R. (M.D. Thesis, Ain-Shams University, 1978) who found that pathologic changes of bone in CRF is uncommon and almost always mild, even among those with advanced CRF. Rehan group (El-Maady Hospital, 1981) however, reported high prevelance rate.

Hormonal assay of parathyroid gland is not that common for studied carried in Egypt. Alaa-El-Din, Z, (Master Thesis, Kasr El-Aini, University, 1980) reported that parathyroid hormone (PTH) is elevated in all patients with CRF and there is no correlation between serum PTH and Geatinine, serum calcium or serum phosphorus. The present study was undertaken to evaluate the serum PTH among population with CRF with and without regular haemodialysis (HD) because PTH has been involved in renal osteodystrophy. Ritz, et al. (1974) with atrial to correlate the findings with different parameters which may be involved in calcium homeostasis among the egyptian patients with CRF.

Radioimmunoassay was used in assessing immunoreactive PTH. The method used is double antibody technique as described in immuno-nuclear-corporation protocol (1981).

This work is carried out at EL-Maady Armed Forces Hospital, Department of nephrology and metabolism.

AIM OF WORK

The aim of work is to study the serum level of PTH as well as various parameter of the patient with chronic renal failure with and without regular haemodialysis (HD).

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REVIEW OF LITERATURE

HISTORICAL REVIEW

The association of albuminuria and rickets was noted as early as (1883) by lucas. Mac-Callum (1905) reported a case of chronic glomerulonephritis with one parathyroid gland enlargement and others were normal. Fletcher (1911) recognized the relationship between chronic renal disease and bony deformuties. Hubbard and Wentworth (1921))reported cases of metatatic calcification and osteitis fibrosa in chronic nephritis.

Mitchell (1930), Smyth and Goldman (1934), Pappenheimer and Wilens (1935), Gilmour and Martin (1937) all confirmed parathyroid gland enlargement in renal insufficiency. Castelman and Mallory (1932) descriped three main types of parathyroid disease on the basis of gross and microscopic observation;

(1) Adenoma is charactized by proliferation of any of the cell type in parathyroid gland, chief cells, oxyphil cells and water clear cells. One gland or at most two glands are involved and the remaining parathyroid tissue is normal. adenoma varies from microscopic size to 120 gms. adenoma involves only one part of the gland.

(2) Primary hyperplasia is due to diffuse enlargement in a size or number of the water clear cells, the stimulus here is unknown the kidney is not affected and all the four glands are enlarged.

(3) Secondary hyperparathyroidism secondary to renal insufficiency affects all the glands to an almost equal degree, there is characteristic microscopic appearance in hyperplasia with the cheif cells or principal showing great increase in number.

Albright (1934) and Albright, dark and Sulkowitich (1937) were the first to draw attention to the rare combination of adult renal insufficiency, chronic acidosis and hyperplasia of parathyroid glands. Also in 1969 Potts et al. stated that hyperparathyroidism characterised by an excessive but not autonomous rate of hypersecretion of parathyroid hormone is encountered invariably in chronic renal failure.

For many years, secondary hyperparathyroidism had been postulated to be present in patients with chronic renal failure. It was known from postmortem studies of patients with chronic renal failure that

the parathyroids were hyperplastic, in addition, changes typical to osteitis fibrosa were found in many patients with far advanced renal failure (Stanbury et al. 1968).

Massry et al. (1969) stated that hyperplasia of parathyroid and high levels of circulating parathyroid hormone are common in patients with chronic renal failure and even in those with mild impairement of renal function.

However, the syndrome of overt hyperparathyroidism is not frequently seen in those patients. Massry et al. (1969), Potts et al. (1969) said that with prolongation of life of patients with chronic renal failure, overthyperparathyroidism is being observed with increasing frequency.

Reiss et al. (1969) reported that excessive secretion of parathyroid hormone has been suspected in renal insufficiency on morphologic grounds. Reiss et al. 1968 and Stanbury 1978 found that in most, if not all, patients with chronic renal failure hyperplasia of the parathyroid glands develops to some degree. Freitage et al. (1978) stated that hyperparathyroidism with markedly