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Introduction

Chronic renal failure is a major health problem in Egypt, and the number of patients on regular hemodialysis is increasing nowadays. At the same time, its different lines of therapy (conservative, dialysis, and transplantation) open wide hopes for long term survival of uremic patients.

Increased secretion of parathyroid hormone (PTH) is common in patients with chronic renal failure, and the excess blood levels of PTH could be deleterious to many organ systems [1]. A variety of symptoms and signs including encephalopathy, neuropathy, dialysis dementia, reduced left ventricular ejection fraction, impaired insulin secretion, glucose intolerance, hyperlipidaemia, soft tissue calcification, bone resorption, pruritis, immunological disturbances, and sexual dysfunction are encountered in patients with uremia[2]. These manifestations contribute to the uremic syndrome and at least part of their pathogenesis has been attributed to PTH as a uremic toxin, and it is thought that such actions of PTH are mediated by an increase in intracellular calcium [2].

Uremic patients are thought to be insulin resistant and about half of them were found to have impaired glucose tolerance [3]. The insulin resistance is a characteristic feature of uremic patients; a problem that could be partially ameliorated both by hemodialysis and by the chronic ambulatory peritoneal dialysis [4,5].

Some evidences suggest a role for PTH in the pathogenesis of deranged beta cell function of uremia. Early studies reported that PTH has a role in the problem of insulin resistance [6]. Subsequent investigations using clamp techniques demonstrated that the development of glucose intolerance in uremic dogs could be prevented by parathyroidectomy [7]. Administration of PTH to rats with normal renal function increased the resting cytosolic calcium, decreased the ATP content and impaired insulin release through ATP-dependent K-channels[8]. Pancreatic islets are apparently a PTH target; PTH stimulates cAMP production and possibly also protein kinase C in a calcium-dependent fashion [9]. Also, it was suggested that uremic patients with elevated serum PTH were found to be severely insulin resistant and hyperinsulinemic and intravenous vitamin D treatment led to a significant reduction of serum PTH levels and to complete normalization of insulin sensitivity in hemodialysis patients [10].

However, the exact effect of hyper-parathyroidism on different parameters of glucose homeostasis, and the effect of lowering serum PTH by one alpha hydroxycholecalciferol therapy still need further evaluation. It is theoretically possible that the deficiency of 1,25 $(OH)_2D_3$ commonly encountered in patients with CRF plays a role in the impaired insulin secretion encountered in these patients[11].

The aim of the study is to look into the relation between serum PTH and parameters of glucose homeostasis and to analyze the effect of lowering PTH on these parameters.

Materials and Methods

The present study was conducted on forty one subjects suffering from chronic renal failure due to different intrinsic renal causes