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Assessment of Calcium Phosphorus and Parathyroid Hormone in Sudanese Patient with Renal Transplantation

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ABSTRACT

Kidney transplantation is the treatment of choice for end-stage renal disease (ESRD). Restoration of renal function after kidney transplantation rectifies many of the disturbances that lead to renal osteodystrophy, bone disease and cardiovascular disease and an increased risk of morbidity and mortality. Identifying patients at risk and evaluating for this complication is imperative because early intervention may slow or arrest the progression of both bone and cardiac disease. Helping physician and renal transplant center to creating a successful treatment plan. The objective of this study was to evaluate calcium, phosphorus and parathyroid hormone concentrations in renaltransplantation in Sudanese patient. This study was case control hospital based study, conducted in Sudanese renal transplantation association, Khartoum state. During June to July 2013.The study involved a control group (n= 50) matched for age with a renaltransplantation patient (n= 50). The ages ranged in both groups were 20 to 65 years. Serum calcium and phosphorus was measured by spectrophotometric methods using chemistry analyzer (cobas c311 analyzer, Germany). While Parathyroid hormone (PTH) levels were determined by electrochemiluminescence using hormone analyzer (cobas e411 analyzer, Germany). The (Mean±SD) of Ca, PTH and posphorus in patient respectively were (9.87±0.52mg/dl, 56.45±26.21 pg/ml,3.36±0.55 mg/dl).While (Mean±SD) of control group was (9.76±0.59mg/dl, 56.02±15 pg/ml,42.3.66±0.43 mg/dl).There was significant decrease of serum phosphorus (p0.003) in subjects with ESRD while Ca and PTH showed no significant difference. This study concluded that, ESRD significant decrease of serum phosphorus, while Ca and PTH were normal in the study.

Key words: Calcium, Phosphorus, PTH, Renal failure, Sudan.

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INTRODUCTION

Kidney transplantation is the treatment of choice for end-stage renal disease (ESRD). Restoration of renal function after kidney transplantation rectifies many of the disturbances that lead to renal osteodystrophy. There is resolution of hypophosphatemia, an increase in serum 1,25 (OH) 2D levels and a rapid decline in the elevated levels of parathyroid hormone (PTH), although they may never completely normalize. In the first few months after kidney transplantation, PTH-dependent hypercalcemia and hypophosphatemia may develop. In addition, post-transplant hypophosphatemia may also be related to a primary defect in renal phosphate handling due to increased serum concentrations of circulating factors (Green et al., 2001). Bone loss rates are greatest in the first 6 to 18 months after kidney transplantation and range from 5 to 8% at the hip and 4 to 9% at the lumbar spine (Ebeling, 2009). Bone loss has not been consistently related to gender, patient age rejection episodes, activity level, or PTH levels. Fracture prevalence varies from 7 to 11% in non-diabetic renal transplant recipients, but is considerably higher in patients transplanted because of diabetic nephropathy and in those who receive kidney-pancreas transplants (Nowacka-Cieciura et al., 2006).

Table 1. Biochemical parameter in patients with renal transplantation.

Parameters	Patient (n =50)	Control (n=50)	P Value
Parathyroid hormone pg/ml	56.45±26.21	56.02±15.42	0.919
Ca mg/dl	9.87±0.52	9.76±0.59	0.307
Phosphorus mg/dl	3.36±0.55*	3.66±0.43*	0.003

*, significant difference.

Fractures occur relatively late in the post-transplant period, usually within the first three years after transplantation, and more commonly involve appendicular sites (hips, long bones, ankles and feet) than the axial sites (spine and ribs) (Nisbeth et al., 1999). A large study of ESRD patients demonstrated that kidney transplantation was associated with a 34% greater risk of hip fracture than continued dialysis (Ball et al., 2002). The importance of phosphorus is exemplified by its distribution within body. For a 70-kg man, there is approximately 23,000 mmol (712 gm) of phosphorus (Knochel, 1977). Hypophosphatemia is an electrolyte disturbance commonly found in early post-transplantation period (Herdman et al., 1966; Hampers et al., 1969; Higgins et al., 1990; Steiner et al., 1993; Ambuhl et al., 1999; Caravaca et al., 1998) persistent hypophosphatemia has been implicated in the development of osteomalacia in those with (Moorhead et al., 1974) and without renal transplant (Bloom and Flinchum, 1960; Lotz et al., 1964). This study was conducted to evaluate calcium, phosphorus and parathyroid hormone concentrations in Sudanese patient with renal transplantation.

MATERIALS AND METHODS

Study Population

This study was Case control descriptive study Hospital based In Sudanese renal transplantation association, Khartoum state. The study during June to July 2013. 50 patients with renal transplantation (38 male, and 12 female) the age ranged (20 to 65 years) the duration of renal transplantation was (7 month to 20 years). 50 persons as control group from Khartoum Teaching hospital Khartoum Sudan (38 male and 12 female) the age ranged (20 to 65 years). Serum Calcium, Phosphorus, measured using specrtophotometric methods. While Parathyroid hormone levels were determined by electrochemiluminescence using hormone analyzer (cobas e411 analyzer - Germany).

Inclusion and Exclusion Criteria

Sudanese patients under goes transplantation for at least 7 month to 20 years. While exclusion patient suffering from parathyroid gland damage, tumors or removal, autoimmune disease, low blood magnesium and vitamins deficiency.

Blood Sample and Data Collection

5 ml venous blood samples were collected from each volunteer the blood samples were centrifuged at 3000 rpm for 10 min, aliquotted, and stored at -20°C until analysis.

Ethical Approval

We obtained ethical approval to carry this study from the ethical committee of Faculty of Medical Lab Science Alneelain University.

Data Analysis

Statistical evaluation was performed using the Microsoft Office Excel (Microsoft Office Excel for windows; 2007) and SPSS (SPSS for windows version 19). Normal distribution of the studied variables was examined using Kolmogorov-Smirnova and Shapiro-Wilk tests. Unpaired T-test and Mann-Whitney U test were used to assess significant difference in the means of the studied variables in renal transplantation patients and healthy individuals. Correlations between serum biochemical profile and the duration of transplantation were assessed using bivariate correlations. $P < 0.05$ was considered statistically significant.

RESULTS AND DISCUSSION

This study was carried out on 50 renal transplant patients (cases) and 50 apparently healthy individual (control) to determine the effect of renal transplantation on parathyroid hormone level, calcium and phosphorus Table 1. The results obtained show that there is no statistically significant difference in levels of calcium, in the renal transplant patients (cases) compared with apparently healthy individuals (control), also cases show no statistically significant difference in levels of parathyroid hormone compared with apparently healthy individuals (control), also the results showed significant decrease in the levels of phosphorus this indicates that

renal transplantation have effect on the levels of phosphorus (hypophspohotimia). These results agree with study reported that in the first few months after kidney transplantation, PTH-dependent hypercalcemia and hypophosphatemia may develop (Green et al., 2001). In addition, post-transplant hypophosphatemia may also be related to a primary defect in renal phosphate handling due to increased serum concentrations of circulating factors (Green et al., 2001). Hypophosphatemia related to decrease renal tubular reabsorption is a common complication following kidney transplantation, and is usually limited to the early post-transplant period (Ambuhl et al., 1999; Ghanekar et al., 2006). However, in some cases, hypophosphatemia persists for more than 10 years after transplantation (Felsenfeld et al., 1986) persistently elevated parathyroid hormone (PTH) levels have long been considered to be the cause of post-transplant hypophosphatemia, but hyperparathyroidism does not appear to be the only mechanism. Decreased renal tubular reabsorption may occur, despite low levels of PTH, and hypophosphatemia can persist even after elevated PTH levels have normalized (Green et al., 2001; Parfitt et al., 1986; Rosenbaum et al., 1981; Graf et al., 1979).

There was a significant difference in serum calcium level in patients with a high serum PTH, as compared to those with a normal iPTH. No significant difference in serum phosphorus was noted between the two groups of patients. This is most probably attributed to factors other than phosphaturic effect of PTH, which results in a low phosphate level immediately after transplantation. An initially low concentration of phosphate after transplantation is due to decreased phosphate reabsorption from the renal tubule (Rosenbaum et al., 1981; Parfitt et al., 1986; Reinhardt et al., 1998) and the phosphaturic effect of steroid, which gets better with tapering the steroid. We observed a gradual rise, from a low serum phosphate concentration up to one year after renal transplantation. In Dousdampanis P, Trigka K, Fourtounas C, Vlachojannis JG study, phosphorus levels decreased significantly after 3 months post-Tx. Hypophosphatemia is a common complication of renal transplantation. Recently, fibroblast growth factor 23 (FGF 23) emerged as its most important mediator, as increased FGF 23 levels, but not PTH levels are independently associated with low serum phosphorus in renal transplant recipients (Hampers et al.,1969). However, increased PTH may act synergistically to increase phosphaturia in these patients (Evenepoel et al., 2000). While Dr. Tong Mei WaGensy study reported that Serum phosphate level falls, not uncommonly to really low level (<0.32 mmol/l), after successful renal transplantation. Re-distribution of phosphate between intracellular and extracellular compartments, osmotic diuresis and renal phosphate loss under the influence of high parathyroid hormone level are the main mechanisms for the development of hypophosphatemia in early post-transplantation period. The short-term and long-term consequences of post-transplantation hypophosphatemia are unconfirmed.

CONCLUSION

This study conducted that, significant decrease of serum phosphorus, while Ca and PTH were normal in the study trying to avoid the acute complications and late effects of hypophosphatemia by given the patient sufficient dosage of phosphate replacement in renal transplant centers.

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