Introduction

Hemodialysis patients are characterized by a number of biochemical abnormalities including hyperlipidemia. The importance of cardiovascular illness as the cause of death in hemodialysis patients, make it imperative to consider the risk factors involved (2). Hyperlipidemia has been incriminated as a risk factor of atherosclerotic vascular disease in dialyzed patients (6). Hemodialysis is associated with hypertriglyceridemia without cholesterol accumulation. The principle other dyslipidemias consisting of high serum lipoprotein (a) levels and low serum high density lipoprotein, whereas plasma low density lipoprotein (LDL) cholesterol is usually not elevated (2,6,7). The cause of hypertriglyceridemia is an increased production of Apo B protein and a marked decrease in the metabolism of VLDL. Primarily as a result of decreased endothelial cell delipidation of VLDL (6). Magnesium (Mg) retention can be a problem in patients on maintenance hemodialysis. Magnesium deficiency has a possible role in the regulation of some enzymes responsible for lipoprotein synthesis. Correlation of serum magnesium with serum triglycerides can be due to changes in hepatic triglyceride metabolism. Lipoprotein(a) is a non-traditional factor of premature atherosclerosis, its association with serum magnesium needs more attention in hemodialysis patients.

Materials and methods

This study is descriptive-analytic that was carried out on thirty-six patients under regular hemodialysis due to end-stage renal failure disease. Factors served as exclusion criteria were antilipid drug taking and active or chronic infection. For patients' plasma cholesterol (Chol) Triglyceride (Tg), High density lipoprotein-cholesterol (HDL-C), Lipoprotein (a) [Lp(a)] and Intact parathormone (iPTH), serum Magnesium (Mg), Calcium (Ca), Phosphorus(P) were measured. Low-density lipoprotein-cholesterol (LDL-C) was calculated by Friedewald’s formula (3). Lipoprotein (a) measured by enzyme immunoassay (ELISA) with Immuno-biological laboratories (IBL) kit of Germany, other lipids were measured by standard kits, iPTH was measured by RIA with DSL-8000.
Results

The total patients were thirty-six (F=16 M=20). The mean ± SD of age of patients were 47.5 ± 16.6 years. The length of the time patients have been on hemodialysis were 25 ± 24.4 months. Table one shows patients data. In this study, there was a significant positive correlation between serum Magnesium and Lipoprotein(a) (r=0.541, p<0.01) (Fig. 1). Significant positive correlation between serum Magnesium with Triglyceride level (r=0.368, p=0.014) was observed too, (Fig. 2). There were not significant correlation between serum Magnesium with Cholesterol, HDL-C, and LDL-C (p>0.05). There were not any positive correlation between serum Calcium, iPTH and Ca x P product with serum lipids (P>0.05).

Discussion

In this study there was a positive correlation between serum Mg levels and serum Lp(a) also significant positive correlation of serum Mg levels with Tg was seen too, means high serum Mg in hemodialysis patients might be associated with some types of dyslipidemia seen in hemodialysis patients. Uremic patients undergoing hemodialysis had dyslipidemia consisting of high serum Tg and Lp(a) levels without cholesterol increment, while High-density lipoprotein-cholesterol has generally been found to be decreased (1,2,6). There is well documented that this lipid profile especially high serum Lp(a) and low HDL-C are highly atherogenic and are one of factors that accelerate atherosclerosis seen in these patients (1,2,3,6,7,11) and needs more attention to find the etiology and treatment of this dyslipidemia.

It has been suggested that Mg deficiency is related to alteration in lipid metabolism (11). Some animal studies showed that Mg-deficient diet is associated with high serum Tg or Cholesterol levels. Rayssiguier et al. showed that in Mg-deficient non-uremic rats, no changes in serum cholesterol were found (9,10). In this way Inagaki et al. found in uremic rats, Magnesium deficiency increased Tg levels and decreased HDL-chol levels (4). In contrast Itoh et al. utilized magnesium hydroxide (548 mg/dl to males and 411 mg/dl for females) for 4 weeks and noted a significant improvement in blood pressure, additionally, the HDL : LDL ratio and total cholesterol improved with the magnesium treatment (5). Recently Robles et al. in a study on twenty-five hemodialysis patients found a positive significant correlation between serum magnesium levels and total cholesterol, and a nearly significant correlation between serum Tg and serum Triglycerides (11). In contrast to this study we found no positive correlation between serum cholesterol with magnesium levels. We found a positive correlation between

Tab. 1: The results of laboratory data.

| Variable  | Maximum | Minimum | Mean ± SD  
|-----------|---------|---------|------------
| LP(a) µmol/L | 3.4     | 0.32    | 1.3±0.70   |
| Tg mmol/L   | 3.4     | 0.45    | 1.53±0.75  |
| Chol mmol/L | 5.8     | 2.6     | 3.98±0.8   |
| HDL-C mmol/L | 1.3     | 0.47    | 0.74±0.26  |
| LDL-C mmol/L | 4.14    | 0.26    | 2.3±1      |
| iPTH ng/L   | 2234    | 25      | 439.4±433.3|
| Mg mmol/L   | 1.65    | 0.29    | 0.6±0.3    |
| CaXP       | 95      | 30      | 54.2±16.6  |

Fig. 1: Correlation of serum Magnesium with serum Lp(a) values (r=0.541, p<0.01).

Fig. 2: Correlation of serum Magnesium with serum Triglyceride values (r=0.368, p=0.014).
serum magnesium and triglyceride too. Lipoprotein(a) is an independent risk factor for atherosclerotic cardiovascular disease (CVD) in general population and dialysis patients (7). In this study we found a significant correlation between serum Magnesium and LP(a) levels. Serum Lp (a) elevation in hemodialysis patients can be due to uremia that could influence Lp(a) metabolism. The kidneys may play a role in Lp(a) catabolism and the end-stage renal disease might result in elevated Lp(a) levels (1,6,8,12). Based on recent observations, showing significant correlations between serum lipoprotein (a), IL-6 and TNF-α. Levels, it is hypothesized that an activated acute phase reaction may be the underlying cause for high levels of lipoprotein (a) found in patients receiving chronic hemodialysis (5,11). Magnesium does not seem to increase lipoprotein synthesis. It may be involved in the regulation of some enzymes responsible for lipoprotein synthesis (11). While there were a trend toward an increase of triglycerid levels with increasing Magnesium levels it could be due to changes in hepatic triglycerid metabolism induced by Magnesium(11). In the meantime, further clinical study into this important aspect of hemodialysis patients is needed.

References