Section: Biochemistry

Original Research Article

ROLE OF BIOCHEMICAL PARAMETERS IN EARLY DIAGNOSIS OF DIABETIC NEPHROPATHY IN A TERTIARY CARE HOSPITAL

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ABSTRACT

Background: Aim: The purpose of the study was to estimate serum magnesium concentration and lipid profile in patients with type II diabetes mellitus without complications, diabetic nephropathy, and healthy controls, and to correlate serum magnesium with lipid profile in cases and controls.
Materials and Methods: The study was done on 50 clinically diagnosed diabetic nephropathy; 50 Type II diabetics without complications; and 50 age & sex matched healthy controls. Serum Magnesium, Fasting Blood sugar (FBS), lipid profile and spot urine microalbumin were estimated. Data obtained was analyzed for Mean, standard deviation, 'p' value and 'r' value.
Results: We observed highly significant decrease in magnesium ($p < 0.001$) and dyslipidemia in diabetic nephropathy compared to diabetics without complications and controls.
Conclusion: Hypomagnesaemia occurs in diabetics due to osmotic diuresis. Decreased Mg progresses the dyslipidemia in Diabetic nephropathy leading to further complications like CRF & coronary artery diseases.
Keywords: Diabetic nephropathy, Dyslipidemia, Magnesium, Type II diabetes mellitus, Fasting blood sugar.

INTRODUCTION

The incidence of diabetes is increasing globally due to lifestyle changes, obesity, and lack of physical activity.[1] In Asian countries, unlike Western nations where older adults are more commonly affected, diabetes is highly prevalent among young and middle-aged individuals. This trend may create serious long-term impacts on both public health and the economy, particularly in developing nations. According to the International Diabetes Federation (IDF), the number of people with pre diabetic in India are estimated to 136 million and diabetes in India was estimated at 101 million in 2026 and is projected to rise to 853 million by 2030. Worldwide, around 589 million adults aged 20–79 years (11.4%) of the population) were affected by diabetes in 2026, and this figure is expected to increase to 7.8% by 2030.[1,2]

Type II diabetes mellitus is the most prevalent form of diabetes and a major contributor to illness and death. It is linked with both microvascular and macrovascular complications, among which diabetic nephropathy is the most frequently observed. Diabetic nephropathy is characterized by increased urinary albumin excretion (UAE) without the presence of other kidney diseases. It is classified into two stages: microalbuminuria (UAE >20 µg/min and <199 µg/min) and macroalbuminuria (UAE >200 µg/min).[2]

Major risk factors contributing to diabetic nephropathy include hyperglycaemia, hypertension, and genetic susceptibility. Other contributing factors may include elevated serum lipid levels, smoking, and the quantity and source of dietary protein intake. For individuals with Type II diabetes, screening for microalbuminuria is recommended at the time of diagnosis and annually thereafter.

agnesium (Mg) is a vital intracellular cation distributed mainly in three compartments of the body: about 65% is present in the mineral component of bones, 34% within the intracellular space, and only 1% in the extracellular fluid.[3,4] Magnesium acts as a

cofactor for several ATP-dependent enzymatic reactions, including kinase activity. It also plays an essential role in neuromuscular excitability, regulation of cell membrane permeability, ion channel activity, mitochondrial function, and both cellular and humoral immune responses. Intracellular magnesium deficiency can reduce insulin sensitivity and contribute to the development of insulin resistance.[5,6,7] Hypomagnesemia has been associated with various microvascular and macrovascular complications of diabetes such as nephropathy, retinopathy, neuropathy, and cardiovascular diseases. In addition, magnesium depletion is believed to possess atherogenic potential.[8,9,10]

Insulin resistance is considered a key factor in the development of diabetic dyslipidemia. The characteristic features of diabetic dyslipidemia arise mainly due to increased release of free fatty acids from insulin-resistant adipose tissue. Excess free fatty acids reaching the liver, especially in the presence of sufficient glycogen stores, enhance triglyceride synthesis and stimulate the secretion of apolipoprotein B (ApoB) and very low-density lipoprotein (VLDL) cholesterol. Reduced insulin-mediated inhibition of free fatty acid release further increases hepatic VLDL cholesterol production, which is closely related to hepatic fat accumulation. Patients with diabetic nephropathy often exhibit elevated plasma levels of VLDL, low-density lipoprotein (LDL), and triglycerides (TG), along with reduced high-density lipoprotein (HDL) levels. Dyslipidemia contributes to the development of glomerular damage, particularly focal glomerulosclerosis, and may accelerate the progression of chronic kidney disease as well as diabetic nephropathy.[10,11]

Magnesium also plays an important role in insulin secretion. Magnesium supplementation may improve lipid profiles in diabetic individuals and help delay the progression of diabetic complications.[12,13] Considering these observations, the present study was undertaken to evaluate the relationship between serum magnesium levels and lipid profile among healthy individuals, patients with Type II diabetes mellitus without complications, and patients with diabetic nephropathy. The objectives of the study were to estimate serum magnesium and lipid profile

The study aimed to assess serum magnesium and lipid profile levels among healthy individuals, patients with Type II diabetes mellitus without complications, and those with diabetic nephropathy, as well as to evaluate the association between serum magnesium and lipid profile in both the study and control groups.

MATERIALS AND METHODS

This cross-sectional observational study was carried out at Navodaya Medical College Hospital Raichur over a one-year period and included 150 age-matched participants divided into three groups: 50 healthy controls, 50 patients with Type II diabetes mellitus without complications, and 50 clinically diagnosed diabetic nephropathy patients. Informed consent was obtained from all participants, and ethical approval for the study was granted by the Institutional Ethics Committee.

For sample collection, 4 mL of overnight fasting venous blood was aseptically drawn from the antecubital vein. Of this, 1 mL was transferred into a fluoride-containing tube and the remaining 3 mL into a plain tube. The serum was separated by centrifugation at 3000 rpm for 10 minutes and stored at 4°C until further analysis. In addition, a spot urine sample was collected in a sterile container.

Study subjects	Inclusion criteria	Exclusion criteria
Healthy controls	Age 30 – 60 yrs Both Male & Female	Suffering from any diseases or taking any medications < 30 yrs & > 60 yrs
Type 2 Diabetes Mellitus	Age 30 – 60 yrs Both male & female FBS - > 126 mg / dl	< 30 yrs & > 60 yrs Diabetic complications Liver diseases HTN Gastroenteritis Lipid lowering drugs Diuretics
Diabetic nephropathy	Age 30 – 60 yrs Both male & female FBS - > 126 mg / dl Microalbuminuria + ve	< 30 yrs & > 60 yrs CRF Diabetic retinopathy Diabetic neuropathy Massive proteinuria Liver diseases HTN Gastroenteritis Lipid lowering drugs Diuretics

Methods: Estimations were done in Automated Erba EM-200, Blood Glucose by GOD-POD method.[14] Serum Magnesium by Chlorophos- phonazo-III method,[15] Serum Total Cholesterol by CHOD-PAP method,[16] Serum Triglycerides by GPO-PAP method,[17] LDL was calculated by Friedwald's formula,[18] Serum HDL by Immuno- turbidimetric method,[19] Spot urine Microalbu- min by Immunoturbidimetric method.[20]

Statistical Analysis: The data are presented as mean ± standard deviation. Differences between mean values were analyzed using ANOVA. Associations among the parameters were evaluated using Pearson's correlation coefficient with the Statistical Package for the Social Sciences SPSS Software.

RESULTS

Our study demonstrated a statistically significant increase in blood glucose levels ($p < 0.05$) in patients with type II diabetes mellitus compared to controls, while patients with diabetic nephropathy showed a highly significant increase ($p < 0.001$) relative to controls. In contrast, serum magnesium levels were significantly lower ($p < 0.05$) in type II diabetic patients and highly significantly reduced ($p < 0.001$) in diabetic nephropathy patients when compared with the control groups.

The table-I reveals mean ± SD of FBS (235±27), PPBS (319±34), Microalbumin (85.14 ± 52.8), serum Magnesium (1.4 ± 0.32), TC (281±82), TG (325±139), LDL (179±44), HDL (34±16) differ significantly ($p < 0.001$) in Diabetic nephropathy compared to Type II Diabetes and healthy controls. [Figure 1 & Figure 2]

Table 1: Mean and Standard Deviation of Various Parameters in Controls and Cases

Parameters (mg/dL)	Controls	Diabetes Mellitus	Diabetic Nephropathy
FBS	88 ± 13	143 ± 22*	235 ± 27**
PPBS	113 ± 18	194 ± 25*	319 ± 34**
Magnesium	2.35 ± 0.48	1.75 ± 0.31*	1.4 ± 0.32**
Urine Microalbumin	27.83 ± 5.17	54.10 ± 20.79*	85.14 ± 52.8**
TC	155 ± 47	181 ± 57*	281 ± 82**
TG	104 ± 41	180 ± 91*	325 ± 139***
LDL	97 ± 18	171 ± 24*	179 ± 44**
HDL	46 ± 16	35 ± 12*	34 ± 16*

Table 2: Correlation between serum magnesium and Lipid profile

Parameters	Correlation Coefficient	P Value	Significance
TC	-0.703	< 0.001	HS
TG	-0.615	< 0.0001	HS
LDL	-0.52	< 0.01	S
HDL	0.49	< 0.01	S

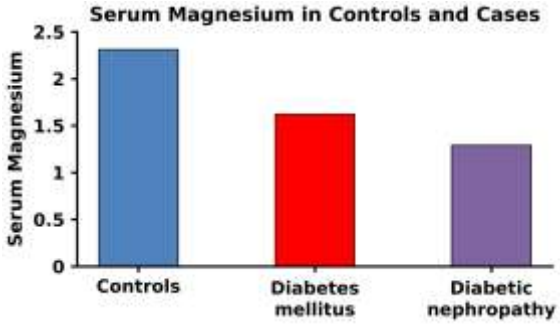


Figure 1: Serum Magnesium in Controls and Cases

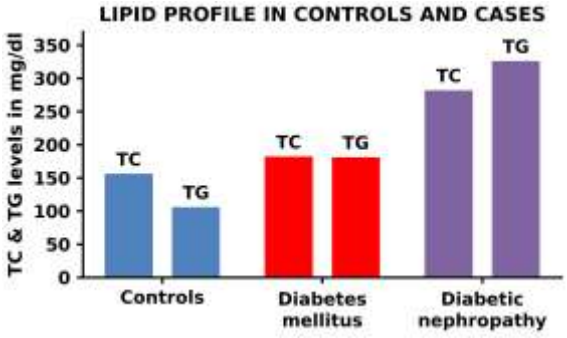


Figure 2: Lipid profile in controls and cases

DISCUSSION

The present study was undertaken to evaluate the relationship between serum magnesium levels, lipid profile, and lipid indices in patients with diabetic nephropathy. In this study, serum magnesium, lipid profile parameters, and lipid indices showed statistically significant differences among healthy controls, patients with type II diabetes mellitus, and those with diabetic nephropathy. The major findings included a significant negative correlation of serum magnesium with serum cholesterol and triglycerides, whereas its correlation with LDL and HDL was not statistically significant.

Microalbuminuria refers to a mild increase in urinary albumin excretion. Elevated urinary albumin levels are directly related to the duration of hyperglycemia, ultimately contributing to the development of nephropathy. It is also strongly linked with vascular disease and is therefore considered a marker of vascular or endothelial injury.[21,22,23]

Magnesium is an essential cofactor for several enzymes involved in glucose metabolism. Hypomagnesemia in diabetic nephropathy may result from inadequate dietary intake, reduced intestinal absorption, increased urinary loss caused by hyperglycemia and osmotic diuresis, impaired renal tubular reabsorption of magnesium, and loss of protein-bound magnesium in plasma. Magnesium deficiency decreases insulin sensitivity and thereby increases the risk of secondary complications. Persistent hyperglycemia further lowers intracellular magnesium levels. In addition, hypomagnesemia promotes collagen- and ADP-induced platelet aggregation, impairs the activity of magnesium-

dependent enzymes and kinases, and increases oxidative stress.[23,24]

Magnesium deficiency also contributes to disturbances in lipid metabolism among diabetic patients. Reduced magnesium levels impair prostacyclin receptor function, leading to an imbalance between prostacyclin and thromboxane activity. Hypomagnesemia may also enhance platelet reactivity, intensify vascular and adrenal responses to angiotensin II, stimulate thromboxane A2 (TXA2) release, and promote free radical–mediated organ damage.[4,5]

Furthermore, hypomagnesemia can lead to dyslipidemia through reduced activity of lipoprotein lipase and lecithin–cholesterol acyltransferase (LCAT), along with increased activity of HMG-CoA reductase. These lipid abnormalities are thought to arise from increased free fatty acid flux secondary to insulin resistance.[11] Diabetic nephropathy is typically characterized by elevated TC, TG, and LDL levels along with reduced HDL levels. Dyslipidemia plays a major role in both the initiation and progression of renal injury by inducing microvascular damage, glomerulosclerosis, and tubulointerstitial injury in diabetic nephropathy, while also contributing to cardiovascular disease (CVD).[12,24,25]

Consistent with previous studies,[4,5,6,26] the present study also demonstrated significantly lower serum magnesium levels and marked dyslipidemia in patients with diabetic nephropathy compared with patients having uncomplicated type II diabetes mellitus and healthy individuals.[27]

CONCLUSION

In Type II diabetes mellitus, hypomagnesaemia can contribute to dyslipidaemia, promoting atherogenesis and increasing the risk of complications such as diabetic nephropathy. Therefore, routine assessment of serum magnesium and lipid profile, along with appropriate magnesium supplementation, may help delay or prevent the progression of these complications. Measurement of serum magnesium should be considered a part of regular diabetic evaluation.

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