ISSN: 0975-833X

Available online at http://www.journalcra.com

International Journal of Current Research
Vol. 12, Issue, 09, pp.13779-13783, September, 2020

DOI: https://doi.org/10.24941/ijcr.39569.09.2020

INTERNATIONAL JOURNAL OF CURRENT RESEARCH

RESEARCH ARTICLE

A STUDY ON EMPLOYEE ENGAGEMENT AND ITS IMPACT ON ORGANISATIONAL EFFECTIVENESS AT WORKPLACE

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ARTICLE INFO

Article History:
Received 19th June, 2020
Received in revised form
17th July, 2020
Accepted 24th August, 2020
Published online 30th September, 2020

Key Words:
Hypomagnesemia; Type 2 Diabetes; Diabetic retinopathy; Albumin creatinine ratio;

ABSTRACT

Background: Diabetic retinopathy irreversibly leads to blindness. Early detection and stringent glycemic control may delay progression but there are no specific biomarkers. Serum magnesium has been found to be associated with retinopathy. **Objective**: To evaluate serum magnesium concentration in patients with type 2 diabetes and to study its association with stages of diabetic retinopathy. **Methods**: Cross section observational study at a tertiary care hospital. Diabetic retinopathy graded as per early treatment diabetic retinopathy study (ETDRS) and diabetes defined as per American diabetic association criteria. Patients with end stage diabetes and those on drugs affecting serum magnesium excluded. **Results**: Patients evaluated after exclusion were 300. The prevalence of hypomagnesemia was 21.3%. The prevalence of retinopathy in hypomagnesemia was 39% and in normal serum magnesium was 17.8%, respectively. The mean serum magnesium levels in type to diabetics without retinopathy, NPDR, diabetic maculopathy, pre-proliferative DR and PDR were 2.1±0.4, 2±0.3, 1.9±0.42, 1.8±0.4 and 1.4±0.07 mg/dl, respectively. Proliferative and pre-proliferative DR was significantly higher in patients with hypomagnesemia. Type 2 diabetics with hypomagnesemia has significantly more ACR>30mg/g than diabetics with normal serum magnesium (Chi-square test, P=0.001). There was a significant and inverse correlation between serum magnesium and ACR (Pearson's correlation coefficient, r= -0.569, P<0.001). There was a significant and inverse correlation between serum magnesium and HbA1c (Pearson's correlation coefficient, r= -0.551, P<0.001). **Conclusion**:Serum magnesium correlates significantly with HBA1c and albumin creatinine ratio and either alone or in combination may be used as a marker for risk and follow-up of patients with diabetic retinopathy.

Citation: ***Varum Malhotra, Hena Dhir, Ankit Daral and Pooja Daral.*** **2020.** "A study on employee engagement and its impact on organisational effectiveness at workplace", *International Journal of Current Research*, 12, (xx), xxx-xx.

INTRODUCTION

Diabetes mellitus is a complex metabolic disorder of carbohydrate and protein and fat metabolism in which there is absolute or relative deficiency of insulin leading to sustained hyperglycemia (Tahseen, 2019). It is estimated that currently about 450 million people are suffering from diabetes, globally, and the prevalence is expected to rise over the next decade. Type 2 diabetes has been found to be associated with low serum magnesium and the prevalence of hypomagnesemia in type 2 diabetics ranges between 14 and 48% as compared to 2.5-15% in healthy controls (Dasgupta, 2012). The normal serum magnesium level ranges between 1.7 to 2.6 mg/dl.

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Serum magnesium concentration less than 1.7 mg/dL constitutes hypomagnesemia and concentrations less than 1.8 mg/dl, pre-clinical hypomagnesemia (Hashizume, 1990; Mazzaferro, 2002). Several clinical trials have shown beneficial effects of magnesium supplementation on glucose metabolism and insulin resistance (Guerrero-Romero, 2014; Guerrero-Romero, 2015). Research has also revealed that hypomagnesemia plays an important role in the pathogenesis of diabetic microangiopathy (Nadler, 1992; Zghoul, 2018).

The World Health Organisation has declared diabetic retinopathy, as the sixth leading cause of blindness, and as an important cause of avoidable blindness.Development of maculopathy and proliferative vitreoretinopathy leads to decrease in vision in patients with long standing retinopathy (Dwyer, 1985; World Health Organization, 2020). Despite numerous studies elucidating the role of hypomagnesemia in chronic diabetic complications; the issue of diabetic retinopathy and hypomagnesemia has been addressed poorly among researchers.

It was believed that strict glycaemic control may delay the development of long-term complications of diabetes. This belief does not hold good for diabetic retinopathy (Rajalakshmi, 2016). The rationale behind present study was to study the correlation between serum magnesium levels, and parameters of glycaemic control (glycosylated haemoglobin and urinary albumin) in patients with diabetic retinopathy.

MATERIAL & METHODS

A cross section observational study was conducted at a tertiary care hospital in the subcontinent from January 2018 to December 2019. The trial was approved by the Institutional Review Boards and the local Ethics Committee. A written informed consent was obtained from all patients willing to participate in the study based on Helsinki protocol.

Inclusion criteria: Patients above 18 years of age with type two diabetes participated in the study. The criteria for diagnosis of diabetes was that suggested by the American diabetes association (ADA). [12] According to ADA, one of these criteria should be met for diagnosis. FPG ≥126 mg/dL (7.0 mmol/L) or 2-h PG ≥200 mg/dL (11.1 mmol/L) during OGTT or A1C ≥6.5% (48 mmol/mol) or in a patient with classic symptoms of hyperglycemia or hyperglycemic crisis, a random plasma glucose ≥200 mg/dL (11.1 mmol/L). Fasting was defined as no caloric intake for at least 8. All patients had serum magnesium levels measured. Type 2 diabetics with normal serum magnesium (1.7-2.6 mg/dl) served as controls.

Sample size calculation: Sample size was calculated from web based sample size calculator of the University of British Columbia for unmatched case control studies using the web link: <https://www.stat.ubc.ca/~rollin/stats/ssize/caco.html>. Type I error (α) was set at 0.05 and power was 80%. The estimated sample size was 300. The grading of diabetic retinopathy was done as per the criteria proposed by Early treatment diabetic retinopathy study (ETDRS). Pupils were fully dilated and stereoscopic examination done using +90D lens. Diabetic retinopathy was identified if any characteristic lesion as defined by the ETDRS severity scale was present and confirmed with fluorescein angiography: microaneurysms, haemorrhages, hard exudates, cotton wool spots, intraretinal microvascular abnormalities, venous beading, and retinal new vessels.The level of retinopathy was graded based on the worse eye. According to the ETDRS study, retinopathy grades were non-proliferative diabetic retinopathy (mild, moderate and severe), diabetic maculopathy, pre-proliferative DR, and proliferative diabetic retinopathy (Solomon, 2019).

Exclusion criteria: Patients less than 18 years, with severe renal dysfunction (serum creatinine ≥ 1.7mg/dl), severe hepatic dysfunction, advanced diabetic end disease (ADED), and patients on drugs affecting serum magnesium like diuretics, aminoglycosides, cyclosporin and amphotericin B were excluded from the study.

Biochemical Measurements: Venous blood samples were obtained after at least 8h of overnight fasting. Glycosylated haemoglobin (HbA1c) levels were measured using high-performance liquid chromatography (HLC-73G7, Tosoh, Tokyo, Japan).

Plasma glucose levels were measured using the glucose oxidase method (Roche Diagnostics GmbH, Mannheim, Germany). Determination of Serum Magnesium was done by photometric xylidyl blue method. [14]HbA1c was measured in whole blood using ion-exchange high-performance liquid chromatography (BIo-Rad). Urinary ACR was calculated as the ratio of the urine albumin level over the creatinine level.Urinary ACR was divided into 4 groups for analysis: less than 10 mg/g, 10 to 30 mg/g, 31 to 300 mg/g, and more than 300 mg/g.

Statistical Analysis: Statistical analysis was performed using IBM statistical software, SPSS Statistics version 26 (IBM Inc.). Data are mean and 95% confidence interval of mean unless otherwise stated. P-value less than 0.05 was considered statistically significant for normally distributedand as medians (interquartile range, IQR) for non-normally distributed continuous variables.Continuous variables were compared using *t*-test. Wherever necessary, the student *t*-test was altered to compare variances that were unequal. The independent-samples t-test was used to determine if a difference existed between the means of two independent groups on a continuous dependent variable. Differences in means (for more than two variables) was calculated using one-way ANOVA in regard to quantitative data and a Kruskal-Wallis-H for non-normally distributed data; and differences in proportions were evaluated using the chi-square test. The association between serum magnesium levels and clinical characteristics was investigated with a Spearman correlation. The association of serum magnesium with retinopathy was assessed with binary logistic regression.

RESULTS

Three hundred and forty type 2 diabetic patients were recruited in the study. Out of these, 20 patients with uncontrolled diabetes and12 patients with advanced diabetic end disease (ADED) were excluded from the study. Eighteen patients refused to take part in the study. The total number of valid cases were 300. The mean age of patients was 61.3±5.2 (range, 50-68 years). There were 200(66.7%) males with a male female ratio of 2:1. The prevalence of hypomagnesemia was 21.3%. The mean age in hypomagnesemia patients was 60.5±4.9 years and in patients with normal serum magnesium was 61.5±5.2 years, respectively (independent t-test, P=0.145). The difference in gender between diabetics with hypomagnesemia and normal serum magnesium was not statistically significant (Chi-square test, P=0.921). The mean serum creatinine in patients with hypomagnesemia was 1.2±0.1and in patients with normal serum magnesium was 0.8±0.1mg/dl (independent t-test, P=0.001). The mean glycosylated haemoglobin in hypomagnesemia was 7.7±0.74% and in normal serum magnesium was 6.4±0.4%, respectively (independent t-test, P=0.001). The mean ACR in hypomagnesemia was 231.2±157 and in normal serum magnesium was 22.4±24.8 (independent t-test, P=0.001). The mean serum magnesium levels in type to diabetics without retinopathy, NPDR, diabetic maculopathy, pre-proliferative DR and PDR were 2.1`±0.4, 2±0.3, 1.9±0.42, 1.8±0.4 and 1.4±0.07 mg/dl, respectively (Figure 1). The prevalence of retinopathy in hypomagnesemia was 39% and in normal serum magnesium was 17.8%, respectively.

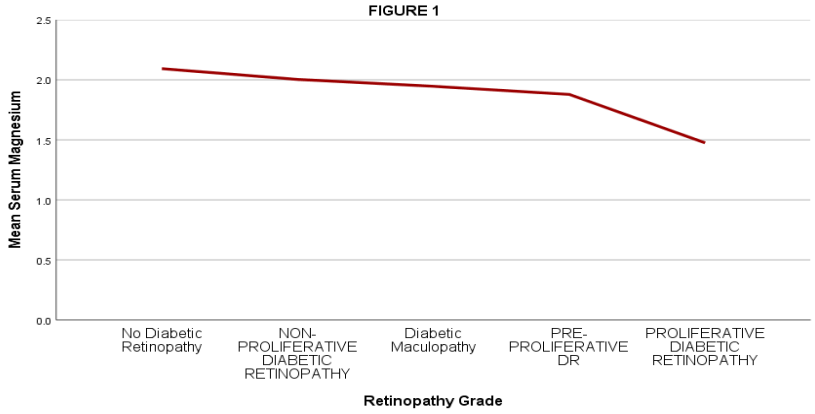

Figure 1. Simple line diagram depicting serum magnesium in patients with and without retinopathy

Table 1. Grade of Retinopathy in Hypomagnesemia

	RETIONPATHY GRADE				
Serum Magnesium	NO DR	NPDR	DM	PRE PROLIF	PDR
Hypomagnesemia	39(60.9)	7(10.9)	4(6.2)	6(9.3)	8(1.2)
Normal magnesium	194(82.2)	25(10.6)	9(3.8)	8(3.4)	0
Total	233(77.6)	32(10.6)	13(4.3)	14(4.7)	8(2.6)

Abbreviations; DR (diabetic retinopathy, NPDR (Non-proliferative diabetic retinopathy, Pre Prolif. (Pre-proliferative DR, PDR (Proliferative diabetic retinopathy).

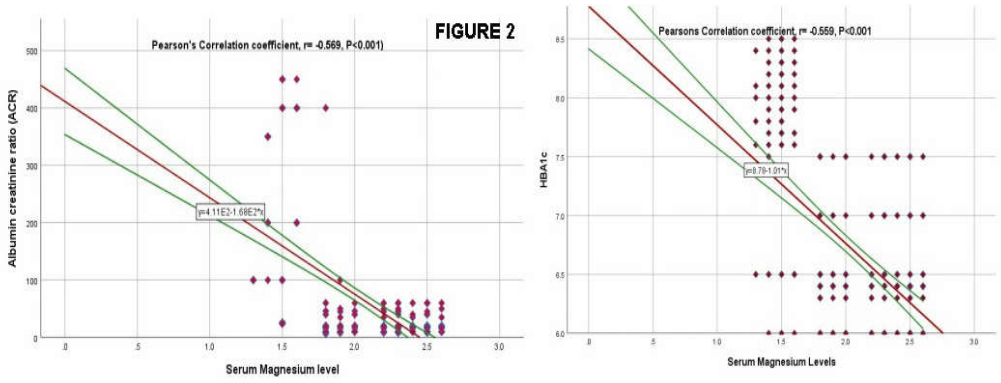

Figure 2. Scatter plot showing correlation between serum magnesium and Albumin creatinine ratio and HbA1c, respectively

Table 1 shows the association between serum magnesium and diabetic retinopathy grade. There was a significant difference in the grade of retinopathy (Chi-square test, P=0.001) between hypomagnesemia and normal serum magnesium. Proliferative and pre-proliferative DR was significantly higher in patients with hypomagnesemia (Figure 1).Type 2 diabetics with hypomagnesemia has significantly more ACR>30mg/g than diabetics with normal serum magnesium (Chi-square test, P=0.001). On correlation analysis, there was a significant and inverse correlation between serum magnesium and ACR (Pearson's correlation coefficient, r= -0.569, P<0.001). There was a significant and inverse correlation between serum magnesium and HbA1c (Pearson's correlation coefficient, r= -0.551, P<0.001).

DISCUSSION

Diabetic retinopathy is one of the leading causes of blindness in the world. Decrease in visual acuity occurs when macula is directly involved or secondary to complications of proliferative retinopathy like vitreous haemorrhage or retinal detachment. Studies have found that intracellular magnesium facilitates the action of insulin by increasing glucose uptake and maintaining vascular tone.

Hypomagnesemia impairs activity of the enzyme tyrosine-kinase, and leads to resistance to the actions of insulin on insulin receptors. The results of the present study revealed that serum magnesium levels were significantly(P<0.05) lower in patients with retinopathy than those without retinopathy; lowest levels of serum magnesium were observed in proliferative diabetic retinopathy (PDR) patients. The exact cause for reduced serum concentration in diabetes still remains unknown. A study by Durak et al found that sustained hyperglycemia may contribute to hypomagnesemia by causing reduced tubular reabsorption of magnesium (Durak, 2010).

The prevalence of hypomagnesemia has shown considerable variation across different studies in the sub-continent. In type 2 diabetics (non-critically ill), prevalence varies between 13.5% to 47.7%. In a case control study by Kumar et al, prevalence of hypomagnesemia was 44%. In our study, the prevalence was 21.3%, which falls within the observed range (Kumar, 2019). In this study we found a significant and direct correlation of glycosylated haemoglobin with the severity of retinopathy and an inverse and significant correlation with reduced serum magnesium.

The role of HbA1c and severity of retinopathy has been evaluated across different studies and relationship has been established with microangiopathy (van den Oever, 2010). Rao et al found that glycosylated haemoglobin and FBS was significantly higher in diabetics with serum magnesium less than 1.7mg/dl. These findings suggest that hypomagnesemia may be a predictor foe early development of diabetic retinopathy. However, this association needs to be further evaluated (Rao, 2015). In the present study we observed a signification association between the grade of retinopathy and extent of hypomagnesemia. Proliferative and pre-proliferative changes were more significantly observed in diabetics with low serum magnesium concentrations. A similar observation was made in a comparative study by Kundu et al in patients with and without retinopathy (Kundu, 2011). Dasgupta et al found that low serum magnesium was associated with microalbuminuria and retinopathy in 64% and 47% type 2 diabetics, respectively.

In our study, 39% patients with hypomagnesemia had retinopathy and 80% patients had ACR>30. Moreover, hypomagnesemiainversely and significantly($P<0.001$) correlated with ACR (Dasgupta, 2012). A similar observation was made by Corsonello *et al*. that diabetic patients with microalbuminuria had a significantly decreased serum ionized magnesium compared with normo-albuminuria group (Corsonello, 2000). The shortcomings of the present study were that duration of disease is also a critical determinant in the type of retinopathy. Second, there was no blinding done giving way to selection bias. Lastly, fundus angiography could not be performed in all study participants due to cost constraints. It is possible that sub-clinical cases could have been missed on stereoscopic examination. In conclusion, microalbuminuria and glycosylated haemoglobin have been indicators of glycaemic control in patients with diabetic retinopathy. Serum magnesium correlated significantly with HBA1c and albumin creatinine ratio and either alone or in combination may be used as a marker for risk and follow-up of patients with diabetic retinopathy.

Acknowledgements: We are thankful to www.indianmedicalstats.com for statistical analysis.

Glossary of Abbreviations: ACR (Albumin creatinine ratio), HbA1c (Glycosylated Haemoglobin), ETDRS (Early treatment Diabetic Retinopathy Study).

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