Study of Serum Magnesium Level in Type 2 Diabetes Mellitus in Relation to Its Microvascular Complication

Dr. Abbas Naqvi¹, Dr. A.K. Shadani², Dr. Shubhangi Gupta³, Dr. I.Rahman⁴, Dr. Pabari Snehaben⁵

Department of Medicine, Ramakrishna Care Hospital, Raipur, India

Corresponding Author: Dr. A.K. Shadani

DOI: https://doi.org/10.52403/ijshr.20220417

ABSTRACT

Magnesium is an essential element and has a fundamental role in carbohydrate metabolism in general and in the insulin action in particular. Magnesium is a cofactor in both glucose transport mechanism of the cell membranes and for various intracellular enzymes involved in carbohydrate oxidation the concentrations of magnesium in serum of healthy people are remarkably constant, whereas 25-39% of diabetics have low concentrations of serum magnesium.

Magnesium depletion has a negative impact on glucose homeostasis and insulin sensitivity in patients with type 2 diabetes, as well as on the evolution of complications such as ischemic heart disease, arterial atherosclerosis, hypercholesterolemia, hypertriglyceridemia, hypertension, retinopathy, neuropathy and nephropathy. Moreover, low serum magnesium is a strong, independent predictor of development of type 2 diabetes.

The study is one and half year observational comparative hospital based study conducted in patients attending the Medicine OPD. Randomly chosen 100 Type 2 diabetic patients, and 100 non diabetic age/sex matched controls were included.

In this study significant correlation is found between Hypomagnesemia and Diabetic Retinopathy. Diabetes Mellitus Type 2 with Hypomagnesemia is associated with more microvascular complications. Serum Magnesium level also found to be correlated with Lipid profile in Diabetics and significant correlation was also found between Hypomagnesemia and Hypertension.

Keywords: Serum Magnesium Level, Type 2 Diabetes Mellitus, Microvascular Complication

INTRODUCTION

Diabetes is on track to become one of the major global health challenges of 21st century. One adult in ten will have diabetes by 2030; figures signify that the number of people living with diabetes is estimated to rise from 366 million in 2011 to 552 million by 2030, if no urgent action is taken. This equates to roughly three new cases every ten seconds or almost ten million per year. ^[1]The chronic hyperglycemia that results may eventually lead to dysfunction of vital organs, especially the heart, kidneys, blood vessels, nerves and eyes. Besides multiplying risk for coronary heart disease, diabetes enhances the incidences of cerebrovascular accidents. Moreover, it is the leading cause of acquired blindness and accounts for about a quarter of the cases with end stage renal disease as well as half of the cases of non-traumatic lower limb amputations.^[2]

The adult human body (approximately 70 kg) contains 21 to 28 gm. of magnesium, which is the fourth most abundant caution in the body and second only to potassium in the cell. Of this about 60% is in bone, 20% in skeletal muscle,

19% in other cells and 1% in extra cellular fluid. It may exist as a protein-bound, complex, or free caution. It plays an role in the carbohydrate important metabolism. It serves as a cofactor for all enzymatic reactions that require kinases.⁽³⁾ Magnesium (Mg) plays a key role in many fundamental biological processes including metabolism and DNA synthesis. Mg deficiency has been shown to cause endothelial cell dysfunction, inflammation, and oxidative stress, which are major contributors to atherosclerosis.⁽⁴⁾

Hypomagnesaemia has long been recognized to be related with diabetes mellitus. In diabetics there is a direct association between serum magnesium level and cellular glucose disposal that is independent of insulin secretion. This change in glucose disposal has been shown to be associated to increased sensitivity of the tissues to insulin in the presence of sufficient magnesium levels. Magnesium deficiency has been found to be related with diabetic micro vascular disease. Magnesium deficiency can lead to development of complications such retinopathy, as thrombosis and hypertension. Several studies have shown that high prevalence of low serum magnesium concentrations in Type 2 diabetes when compared to healthy Hypomagnesaemia has controls. been patients verified in with diabetic retinopathy, with lower magnesium levels predicting a greater risk of severe diabetic retinopathy.⁽⁵⁾

Initially the of cause hypomagnesaemia was attributed to (1) osmotic renal losses from glycosuria (2) decreased intestinal magnesium absorption and redistribution of magnesium from plasma into red blood cells caused by insulin effect. Recently a specific tubular magnesium defect in diabetes has been postulated. Hypomagnesemia results specifically from a reduction in tubular absorption of magnesium.⁽⁶⁾ Mg deficiency is associated with poor glycemic control and supplementation improves Mg insulin sensitivity.⁽⁷⁾



It is observed that low serum magnesium concentration and poor magnesium status are common in Type 2 diabetes Preventing mellitus. hypomagnesaemia in diabetes by supplementing magnesium may be helpful in increasing insulin sensitivity and delaying the development of late diabetic micro vascular complications. Therefore, the aim of the study is to compare the serum magnesium concentrations in patients with Type 2 diabetes and non-diabetic controls to prove hypomagnesaemia is present in Type 2 diabetes mellitus.

MATERIALS & METHODS STUDY SITE: SAMPLE SIZE:

From earlier study [Kulkarni A G, Shendge S K, Shinde V. Study of Serum Magnesium Levels in Types 2 Diabetes Mellitus.IOSR-JDMS.2014 April;13(4):115-119]

of diabetic patients had low serum magnesium levels (Mg2+level<1.5mg/dL) and 9% of controls had low serum magnesium levels (Mg2+level<1.5mg/dL). P1=Proportion of diabetic patient with low

serum magnesium level=37%=0.37

Q1=1-P1=0.63

P2=Proportion of controls with low serum magnesium level =9%=0.09 Q2=1-P2=0.91

1 2

=

Sample size Formula by Fleiss

minimum 33 samples in each group

 $(1.96*\sqrt{2P'Q'}+0.84*(P1Q1+P2Q2)^2)$

 $(P1 - P2)^2$

We took total study sample of 200 in which 100 patient with type 2 diabetes mellitus and 100 controls.

Where $P' = \frac{(P1+P2)}{2} \& Q' = 1-P'$

STUDY DURATION: February 2017 TO July 2018

INCLUSION CRITERIA FOR CASE GROUP:

All cases of Type 2 diabetes mellitus known cases as well as newly diagnosed patients aged between 30 to 70 years attending Medicine OPD, at Ramakrishna Care Hospital Raipur, Chhattisgarh.

EXCLUSION CRITERIA FOR CASE GROUP:

- Patients with renal failure.
- Patients who suffered acute myocardial infarction in last six months
- Patients on diuretics.
- Patients with history of alcohol abuse.
- Patients on magnesium supplements/ magnesium containing antacids.
- Patients with Malabsorption.
- Patient withChronic diarrhea

INCLUSION CRITERIA FOR CONTROL GROUP:

Age and sex matched non-diabetic patients admitted in the hospital were taken as controls after applying the same exclusion criteria which were applied for the cases.

METHODOLOGY AND STATISTICAL METHODS

Source of data:

Randomly chosen 100 Type 2 diabetic patients, and 100 non diabetic age/sex matched controls attending General Medicine OPD at Ram Krishna Care Hospital, Raipur. Between February 2017 to July 2018.

Method of Collection of Data:

Detailed history including Patients age, sex, Duration of diabetes mellitus, Details regarding mode of treatment like using insulin or oral hypoglycemic agents or both, Past history of any Other diseases, History of comorbid diseases like hypertension, Ischemic heart disease, smoking history, usage of alcohol.

Family history of Diabetes and Hypertension taken. Detailed general Physical examination conducted. Pulse, blood Pressure, Respiratory Rate, Temperature, Height, Weight. Body mass index (BMI), which was calculated with the formula weight/

Height² (kg/m²), were recorded for each patient and controls.

A venous blood sample was collected from each subject in the morning after 12-hour fasting, to evaluate fasting glucose and another sample 2 hours after meal for post prandial glucose [hexokinase with enzymatic reference methods] was collected from each cases and controls.

Serum urea and creatinine [kinetic colorimetric assay based on the Jaffe methods] was estimated in each diabetics and healthy controls. HbA1c was done in every diabetic patients. Serum magnesium level from fating venous blood sample was collected [enzymatic end point methods] from each diabetics and normal healthy controls.

For Dyslipidemia Total cholesterol, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), triglyceride (TG), from fasting venous blood sample was taken. Diagnosis of diabetic neuropathy was confirmed by a detailed medical history, and neurological examination. Blood pressure and heart rate with postural change in blood pressure measurement, pinprick sensation test, perception with monofilaments, vibration and position, and assessment of deep tendon reflexes were performed in all diabetic patients.

Diabetic retinopathy was diagnosed with fundus examination.

Biochemical Tests Done Among Diabetic Patients:

- a. FBS and PPBS.
- b. Serum magnesium Levels.
- c. HbA1C.
- d. Lipid profile.
- e. Blood Urea and Serum Creatinine

Biochemical Tests Done Among Non-Diabetic Controls:

- a. FBS and PPBS.
- b. Serum magnesium levels.
- c. Blood Urea and Serum Creatinine.

Method of Estimation Of Serum Magnesium

Principle of the test:

It is an enzymatic end point method. Principle of reaction is that, the Calmagite combines with the magnesium to form a red complex in alkaline solution which is measured at 520 run.

Reagent is in liquid form ready for use.

A magnesium standard is provided with the reagent.

Wave length: 520 nm.

Temperature: 37^oC

Cuvette: 10 nm path length

Incubation: 5 minutes

Standard: 2mg/dL

Kit used in this study was RAICHEM's Magnesium liquid reagent The normal serum magnesium level is ranging from 1.8 mg /Dl to 2.9 mg /dL. Serum magnesium levels </= 1.5 mg / dL is considered as low magnesium level in this study.

Data Summarization - data calculation was done in the terms of MEAN VALUE.

Measures of Variability-Variation Reported in terms of SD

Measures of Probability- confidence interval of 95 % (C.I.) was taken so that it included the true value.

Measures of Significance- P value derived from Z table and took p value <0.05 for significance.

Power of Study- To obtain a statistically significant result in a study 80% of power was taken.

Statistical Analysis

Continuous variable was analyzed by Mean, SD and test of significance by "t" test Categorical data was analyzed using chi square test or fisher exact test.

Statistical software SPSS 16.0 was used for data analysis

Statistical significance

P<0.05 is significant

P<0.01 is highly significant P>0.05 is not significant

LITERATURE REVIEW

Diabetes mellitus, both type-1 and type-2, are among the most common causes deficiency^(8,9,10) magnesium of The incidence of hypomagnesemia in patients with type 2 diabetes ranges widely, from 13.5%-47.7%. Causes include poor oral intake, increased renal loss and the chronic diarrhea associated with autonomic neuropathy. Drugs like proton-pump inhibitors can impair the gastrointestinal absorption of magnesium. This effect may be the result of a drug-induced decrease in the pH of the intestinal lumen that alters the affinity of transient receptor potential melastatin-6 and melastastin-7 (TRPM6, TRPM7) channels on the apical surface of enterocytes for magnesium (8-10).

Probably one of the most studied chronic diseases with respect to magnesium is type 2 diabetes mellitus and the metabolic syndrome. Magnesium plays a crucial role in glucose and insulin metabolism, mainly through its impact on tyrosine kinase activity of the insulin receptor, by transferring the phosphate from ATP to protein. Magnesium may also affect phosphorylate b kinase activity by releasing glucose-1-phosphate from glycogen.

In addition, magnesium may directly affect glucose transporter protein activity 4 (GLUT4), and help to regulate glucose translocation into the cell ^(9,10,11,12).

Recent studies have shown that magnesium intake is inversely associated with the incidence of type 2 diabetes. This finding suggests that increased consumption of magnesium-rich foods such as whole grains, beans, nuts, and green leafy vegetables may reduce the risk of diabetes type 2 ^(13,14,15). A meta-analysis of seven prospective cohort studies from 1966-2007 investigated the association between magnesium intake (from foods only or from foods and supplements combined) and the incidence of type 2 diabetes. 286,668 participants and 10,912 cases were included.

All but one study found an inverse relation between magnesium intake and risk of type 2 diabetes, and in four studies the association was statistically significant. The overall relative risk for a 100 mg magnesium intake per day was 0.85 (95% CI, 0.79–0.92). Results were similar for intake of dietary magnesium (RR, 0.86; 95% CI, 0.77–0.95) and total magnesium (RR, 0.83; 95% CI, 0.77–0.89)⁽¹⁵⁾.

Diabetes is a disease that is strongly associated with both micro vascular and macro vascular complications. Therefore, diabetes is a major public health problem associated with a huge economic burden in developing countries. These complications are wide ranging and are due at least in part to chronic elevation of blood glucose levels, which leads to damage of blood vessels.

Among the most prevalent micro vascular complications are kidney disease, amputations. blindness. and Impaired kidney function, exhibited as a reduced glomerular filtration rate, is also a major risk factor for macro vascular complications, such as heart attacks and strokes. Other chronic complications of diabetes include depression, dementia, and sexual dysfunction ⁽¹⁶⁾.

Magnesium depletion, for example by its effect on inositol transport, is of pathogenic significance in the development of diabetic complications (see Figure 2). A balanced magnesium status is associated with a decreased risk for micro vascular and macro vascular complications ^(17,18,19,20).

According to the recent guidelines of the Association for Magnesium Research. patients with diabetes benefit across four categories from magnesium supplementation: insulin sensitizing effect, calcium antagonism, stress regulating, and endothelium stabilizing effects. In diabetics, the Association for Magnesium Research recommends daily magnesium a supplementation between 240 and 480 mg $(10-20 \text{ mmol})^{(23)}$.



Figure 2: Magnesium deficiency and diabetes (21.22)

RESULT

Estimation of serum magnesium levels of 100 patients of type 2 diabetes mellitus and 100 controls was done in present study.

- Most of the patients belongs to age group of 51-60 years in both diabetics (43%) and control (40%).
- 2. In both the diabetes group percentage of males and females was 59% and 41% while in control it was 54% and 46% respectively.
- 3. According to duration of diabetes most of them were in group of 5-10 Years (44%).
- 4. Mean BMI in diabetics and control was 23.38±3.16 and 22.76±3.38 respectively.
- 5. 66% of patients were receiving oral hypoglycemic agents (OHA), 10% were receiving insulin and 24% were receiving both OHA and insulin for therapy for diabetes.
- The FBS in diabetics ranged from 80 mg/dL to 243 mg/dL with a mean of 124.<u>+</u>27.9mgldL, whereas in controls it ranged from 67 mg/dL to 109 mg/dL with a mean of 89.35<u>+</u>12.81 mg/dL.
- 7. The PPBS in diabetics ranged from 100to 380mgIdl with a mean of 215.65.

 \pm 53.92 mg/dL, whereas in controls it ranged from 96 mg/dL to 138 mg/dL with a mean of 116.2 \pm 11.26 mg/dL.

8. In this study 32% of diabetic patients and 7% of non-diabetic controls had hypomagnesemia. The mean serum magnesium was 1.97 ± 0.52 mg/dl and 2.15 ± 0.34 mg/dl in diabetics and controls respectively.



9. Out of 100 diabetics 23 patients had IHD, 35 patients had Hypertension, 32 patients had Retinopathy and 16 patients had Neuropathy.



10. In diabetics mean serum magnesium level was 1.97 ± 0.52 mg/dL and in non-diabetic controls 2.15 ± 0.34 mg/dL. Mean serum magnesium levels were lower in diabetic patients and the difference was found to be statistically significant with p value 0.0052.



In this study hypomagnesemia (Mg^{2+} level1.5mg/dL) is correlating with Hypertension, hence there is an association between Hypertension and serum Mg2 level and also there is an association between Retinopathy and serum Mg^{2+} level (p=0.024 significant).

11. In this study there is correlation with Lipid profile and serum magnesium level also found. There is positive correlation with HDL and negative correlation with Serum Triglyceride, LDL, VLDL is .



DISCUSSION

Age & Sex Distribution

In the present study age were ranging from 30-70 years. Maximum no of patients was in age group of 41-50 years in both diabetics (43%) and controls (40%). In the diabetes group 59% of the patients were males and 41 % were females. In controls 54 % were males and 46 % were females.

Prevalence OF Hypomagnesemia

Present study showed that 32% of diabetic patients had low serum magnesium levels and 7% of non-diabetic controls had low serum magnesium levels. The mean serum magnesium level was 1.97 ± 0.52 mg/dL and 2.15 ± 0.34 mg/dL in diabetics and controls respectively (p value <0.0001 Highly significant).

Relation between Serum magnesium levels and duration of DM

In the present study, hypomagnesaemia was present in 31.82% patients with duration of 0.5-5 years of type-2 diabetes mellitus, in 30.77% patients with duration of 5-10 years, in 32.56% patients with duration of >10 years. However, there was statistically no significant difference in the prevalence of hypomagnesaemia according to duration of disease (p = 0.91).

Mode of Treatment and hypomagnesemia

In the present study, out of 100 type-2 diabetes mellitus patients, 10% were on insulin, 24% were on oral hypoglycemic agents (OHA)+insulin, while 66% patients were on OHA alone. There was no relation of hypomagnesemia with mode of treatment of type-2 diabetes mellitus (p=0.24), Probably because of the small sample size of the study.

Hypomagnesemia and Diabetic Retinopathy

In the present study, patients with diabetic retinopathy had significantly higher prevalence of hypomagnesemia compared to patients without retinopathy (52.38% vs.

26.58%). The difference was statistically significant (p = 0.024).

Hypomagnesemia and Diabetic Neuropathy

In the present study, 43.75% patients with diabetic neuropathy have low serum magnesium level as compared to 29.76% patients without neuropathy. The differences were statistically not significant (p = 0.27).

Dyslipidemia and Serum Magnesium

In present study a significant rise of Triglycerides (TG), Total Cholesterol (TCh), LDLc and VLDLc in diabetics, in comparison with controls (p<0.001) was found. In cases, the serum magnesium had negative correlation with TG, VLDLc, TCh and LDLc and positive correlation with HDLc was found. This might point out the contributory role of magnesium towards dyslipidemia.

Hypertension and Serum Magnesium

In the present study 54.29% of diabetic patients found to have low serum magnesium which was highly significant p=<0.001. Hence there is with an association between Hypertension and serum Mg²⁺level and it means that patients having low magnesium levels had the risk of developing Hypertension. However, in the present study, there was no scope for follow up so change in magnesium status with respect to improvement or worsening of diabetic status in the long run was also studied here. Also. magnesium not supplementation and its effects towards magnesium levels or metabolic control was not done in this study which can be taken as limitations of the present study.

CONCLUSION

The present study has reconfirmed association of Diabetes Mellitus Type 2 with Hypomagnesemia and also that Hypomagnesemia is associated with more microvascular complications.

In this study significant correlation is found between Hypomagnesemia and Diabetic Retinopathy.

Serum Magnesium level also found to be correlated with Lipid profile in Diabetics and significant correlation was also found between Hypomagnesemia and which Hypertension, may suggest contributory role Magnesium of in Dyslipidemia and Hypertension which are also found to be commonly associated with Diabetes Mellitus.

Whether raising Magnesium level with magnesium supplementation can improve the control of diabetes and reduce or delay the microvascular complications could not be studied because of limitation of time frame in which study was conducted.

However, this is a matter which needs further attention in the form of more research. So further multicentric randomized control study is recommended with larger sample size and proper follow up to strengthen the understanding of this particular problem.

Acknowledgement: None

Conflict of Interest: None

Source of Funding: None

Ethical Approval: Approved

REFERENCES

- 1. H. Kaur, N. Bhaskar, S. Ishaq, Q. Najeeb. Stem Cells: Source for diabetes cell therapy. Journal of Diabetology, October 2012; 3:3
- 2. Hari ML. A study of microalbuminuria in type II diabetes mellitus. Int J Modn Res Revs. 2014; 2(10): 433-37.
- 3. Pham PCT, Pham PMT, Pham SV, Miller JM, Pham PTT. Hypomagnesemia in Patients with Type 2 Diabetes Clin J Am Soc Nephrol 2007; 2: 366–73.
- 4. Wolf FI, Trapani V, Simonacci M, Ferré S, Maier JA. Magnesium deficiency and endothelial dysfunction: Is oxidative stress involved? Magnes Res 2008; 21:58-64.
- 5. Memon AR, Memon MY, Memon F. Serum Magnesium Levels in Type-I and Type-II

Diabetes. Indo American Journal of Pharm Research.2013:3(9): 7029-32.

- Djurhuus MS, Gram J, Peterson PH, Klitgaard NA, Bollerslev J, Beck NH. Insulin increases renal magnesium depletion: a possible cause of magnesium depletion in hyperinsulinemic states. Diabet med 1995;12: 664-669.
- Rodríguez-Morán M, Guerrero-Romero F. Oral magnesium supplementation improves insulin sensitivity and metabolic control in type 2 diabetic subjects: A randomized double-blind controlled trial. Diabetes Care 2003;26:1147-52.
- Palmer B.F., Clegg D.J. Electrolyte and Acid-Base Disturbances in Patients with Diabetes Mellitus. N. Engl. J. Med. 2015; 373:548–559.
- Djurhuus M.S., Skøtt P., Hother-Nielson O., Klitgaard N.A., Beck-Nielsen H. Insulin increases renal magnesium excretion: A possible cause of magnesium depletion in hyperinsulinaemic states. Diabet. Med. 1995; 12:664–669.
- Ramadass S., Basu S., Srinivasan A.R. Serum magnesium levels as an indicator of status of Diabetes Mellitus type 2. Diabetes Metab. Syndr. 2015; 9:42–45.
- 11. Perazella M.A. Proton pump inhibitors and hypomagnesemia: A rare but serious complication. Kidney Int. 2013; 83:553– 556.
- Guerrero-Romero F., Simental-Mendía L.E., Hernández-Ronquillo G., Rodriguez-Morán M. Oral magnesium supplementation improves glycaemic status in subjects with prediabetes and hypomagnesaemia: A double-blind placebo-controlled randomized trial. Diabetes Metab. 2015; 41:202–207.
- Classen H.G., Nowitzki S. The clinical importance of magnesium. 2. The indications for supplementation and therapy. Fortschr. Med. 1990; 10:198–200.
- Forbes J.M., Cooper M.E. Mechanisms of diabetic complications. Physiol. Rev. 2013; 93:137–188.
- Larsson S.C., Wolk A. Magnesium intake and risk of type 2 diabetes: A meta-analysis. J. Intern. Med. 2007; 262:208–214.
- Vormann J. Update: Magnesium und Diabetes. OM-Z. Orthomol. Med. 2014; 1:6–8.
- 17. Mooren F.C., Krüger K., Völker K., Golf S.W., Wadepuhl M., Kraus A. Oral magnesium supplementation reduces insulin

resistance in non-diabetic subjects-A double-blind, placebo-controlled, randomized trial. Diabetes Obes. Metab. 2011; 13:281–284.

- Von Ehrlich B., Wadepuhl M. Erhöhtes Risiko einer diabetischen Retinopathie bei niedrigem Serum-Magnesium. Diabetes Stoffwechs. 2003; 12:285–289.
- De Leeuw I., Engelen W., de Block C., van Gall L. Long term magnesium supplementation influences favourably the natural evolution of neuropathy in Mgdepleted type 1 diabetic patients (T1DM) Mag. Res. 2004;17:109–114.
- 20. Pham P.C., Pham P.M., Pham P.T., Pham S.V., Pham P.A., Pham P.T. The link between lower serum magnesium and kidney function in patients with diabetes mellitus type 2 deserves a closer look. Clin. Nephrol. 2009; 71:375–379.
- Gröber U. Interactions between drugs and micronutrients. Med. Monatsschr. Pharm. 2006; 29:26–35.
- Djurhuus M.S., Skøtt P., Hother-Nielson O., Klitgaard N.A., Beck-Nielsen H. Insulin increases renal magnesium excretion: A possible cause of magnesium depletion in hyperinsulinaemic states. Diabet. Med. 1995; 12:664–669.

- 23. Von Ehrlich E., Barbagallo M., Classen H.G., Guerrero-Romero F., Morren F.C., Rodriguez-Moran M., Vierling W., Vormann J, Kisters K. The significance of magnesium in insulin resistance, metabolic syndrome, and diabetes-Recommendations of the Association of Magnesium Research e.V. Diabetol. Stoffwechs. 2014; 9:96–100.
- 24. Kulkarni A G, Shendge S K, Shinde V.Study of Serum Magnesium Levels in Types 2 Diabetes Mellitus.IOSR-JDMS. 2014 April;13(4):115-119.
- 25. Saproo N, Singh R. Study of serum megnesium levels in diabetes mellitus and its co relation with complications (retinopathy and HbA1c) a cross sectional study of one year. International Journal in Advances of Medicine. 2017 Feb;4(1):263-269.

How to cite this article: Abbas Naqvi, A.K. Shadani, Shubhangi Gupta et.al. Study of serum magnesium level in type 2 diabetes mellitus in relation to its microvascular complication. *International Journal of Science & Healthcare Research.* 2022; 7(2): 108-117. DOI: *https://doi.org/10.52403/ijshr.20220417*
