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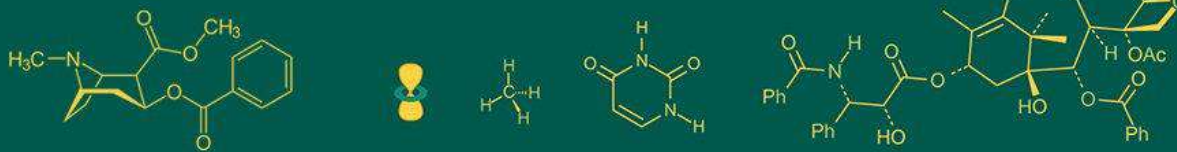
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Study of HbA1c and serum magnesium in diabetic patients with retinopathy

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Abstract
Background: Diabetic retinopathy is one of the main sources of obtained visual deficiency and is a sight-undermining complexity of diabetes mellitus. There is an assortment of danger factors, for example, hypomagnesemia, length of diabetes, and weakened guideline of diabetes, connected to the turn of events and movement of diabetic retinopathy.
Methods: The all-out number of subjects examined was 120, comprising of 40 safe controls and 80 instances of diabetes. Out of 80 instances of diabetes 40 were patients without retinopathy and 40 were patients with retinopathy. HbA1c levels and serum magnesium levels were estimated in diabetic gatherings and the benchmark group. The term diabetes was looked at in diabetic gatherings.
Results: The mean duration of diabetes was higher in the diabetic group with retinopathy when compared to the diabetic group without retinopathy. HbA1c was significantly higher in the diabetic groups when compared to the control group. Serum magnesium was significantly decreased in diabetic groups when compared to the control group. Compared to the diabetic group without retinopathy, the HbA1c was significantly higher and serum magnesium was significantly decreased in the diabetic group with retinopathy.
Conclusion: Increased HbA1c is attributed to poor glycemic control. Decreased serum magnesium levels are attributed to dietary deficiency, impaired magnesium absorption, increased diabetes urination loss. Factors, for example, diabetes mellitus length, glycemic control, and hypomagnesemia influenced the force of diabetic retinopathy in the contemplated gatherings. Early conclusion and treatment in diabetic patients alongside magnesium enhancements can diminish the beginning and movement of retinopathy.

Keywords: Diabetic retinopathy, HbA1c and magnesium

Introduction

Diabetes Mellitus is a complex metabolic infection brought about by hereditary and ecological elements having a variable relationship. It is perhaps the most well-known metabolic issue where either the chemical insulin is missing or the cells of the body are safe from the impacts of insulin [1, 2]. It is associated with a high mortality rate due to several complications that appear to worsen over time and carry a significant risk of premature mortality. Irregular insulin discharge, raised blood glucose levels and various entanglements, for example, retinopathy, nephropathy, neuropathy and arteriosclerosis, are the key attributes [1, 3].

Probably the greatest danger to human wellbeing in the 21st century is Diabetes Mellitus. By 2025, the WHO has anticipated that there will be an ascent to 300 million. The vital variables for the fastest ascent in the commonness of type 2 diabetes mellitus are expanded urbanization and industrialization [4, 5]. Diabetic retinopathy (DR) is an intricacy of diabetes mellitus that undermines sight and is one of the main sources of grown-up procured visual deficiency. Compared to average people, the risk of losing sight is around 25 times higher. There is a range of risk factors linked to diabetic retinopathy development and progression, such as diabetes mellitus duration, poor glycemic control, dyslipidemia, hypertension and hypomagnesemia [1, 2].

It has long been established that hypomagnesemia is linked with diabetes mellitus. It is said that magnesium depletion has a detrimental effect on the homeostasis of glucose and insulin sensitivity. This connection between diabetes mellitus and magnesium has a wide scope of consequences for the guideline of diabetes and its inconveniences [6, 7, 8].

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In developing microangiopathy, chronic hyperglycemia and its associated non-enzymatic glycation play an important role. Hyperglycemia leads to the production of advanced end products for glycation, resulting in numerous vascular complications, such as myocardial infarction, etc. [9] Intensive glycemic control, as measured by serum HbA1c levels, has been shown to reduce diabetic complications, especially microvascular diseases, in randomised trials.

Several studies have been carried out to study the effect on the progression of retinopathy of these individual risk factors. In diabetic patients with retinopathy, however, very few studies have been conducted to research the association between any of these risk factors. During this investigation, an endeavour was made to discover a connection between the beginning and improvement of diabetic retinopathy between these danger factors.

Material and Methods
80 patients with type II diabetes, 40 patients with clinically analyzed retinopathy, and 40 patients without retinopathy were remembered for the examination. The subjects were chosen from the Ophthalmology Department of the outpatient and inpatient units. The clinical diagnosis was based on the results of history and fundoscopy. Patients ranged in age from 30-60 years. The cases were chosen on the basis of a simple method of random sampling. Chronic diarrhoea, alcoholism, breastfeeding, medications that induce hypomagnesemia (such as diuretics, cisplatin, pentamidine), urinary tract infection, inflammatory disorders such as rheumatoid arthritis, myocardial infarction, recent surgical history, and major trauma were included in the exclusion criteria.

After obtaining due consent, the findings were compared with 40 normal healthy randomly selected persons. The controls were balanced by age and sex. Serum magnesium and HbA1c in blood were used in the inquiry. Similarly, the controls were compared.

For the HbA1c calculation, blood obtained in EDTA-containing vacutainers was used. Serum magnesium was estimated in the autoanalyzer using the Xylidyl blue method using RANDOX KIT-MG 3880. HBA1c was estimated by the inhibition method of Latex agglutination using RANDOX KIT-HA 3830 in the HbA1c analyzer.

Results
The age group was between 30-60 years old. The mean age was 49.53 ± 5.82 years for diabetic patients with retinopathy, 46.77 ± 6.99 years for diabetic patients without retinopathy, and 45.50 ± 6.14 years for controls.

Table 1: Comparison of Mean Duration of diabetes in Diabetic cases without retinopathy and Diabetic Cases with retinopathy

Groups		Duration of Diabetes
Diabetic cases without retinopathy	Mean ± S.D	3.64 ± 2.03 yrs
Diabetic cases with retinopathy	Mean ± S.D	9.72 ± 3.64 yrs
Diabetic cases without retinopathy vs Diabetic cases with retinopathy	p-value	<0.001

Contrasted with diabetic patients without retinopathy, the length of diabetes mellitus in diabetic patients with retinopathy was altogether higher ($P<0.001$). The outcomes above demonstrate that as the length of diabetes mellitus builds, the recurrence and improvement of retinopathy can increment.
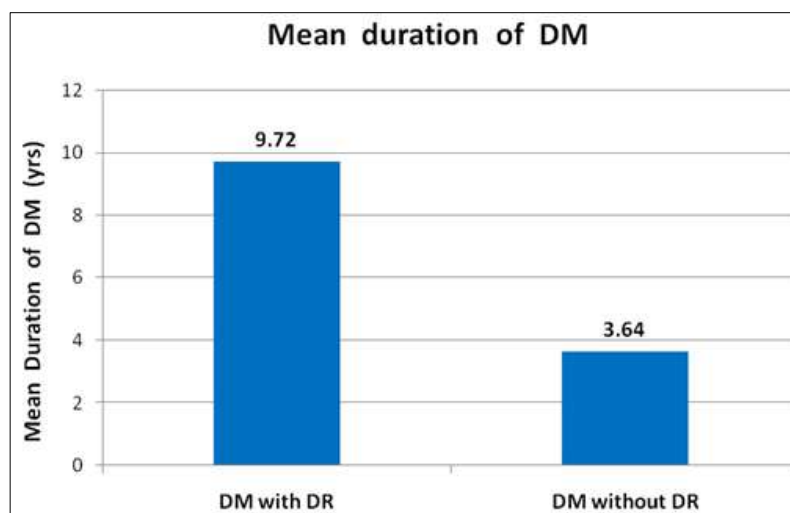

Fig 1: Bar diagram showing the mean duration of diabetes mellitus in the diabetic groups

Table 2: Comparison of Serum Magnesium and HbA1c in Controls and Diabetic cases without retinopathy

Groups		Magnesium (mg/dl)	HbA1c (%)
Controls	Mean ± S.D	2.47 ± 0.24	4.48 ± 0.51
Diabetic cases without retinopathy	Mean ± S.D	2.03 ± 0.19	7.12 ± 0.94
Controls vs. Diabetic cases without retinopathy	p-value	<0.001	<0.001

Table 3: Comparison of Serum Magnesium and HbA1c in Controls and Diabetic cases with retinopathy

Groups		Magnesium (mg/dl)	HbA1c (%)
Controls	Mean ± S.D	2.47 ± 0.24	4.48 ± 0.51
Diabetic cases with retinopathy	Mean ± S.D	1.97 ± 0.35	8.39 ± 1.19
Controls vs. Diabetic cases with retinopathy	p-value	<0.001	<0.001

Table 4: Comparison of Serum Magnesium and HbA1c in Diabetic cases without retinopathy and Diabetic cases with retinopathy

Groups		Magnesium (mg/dl)	HbA1c (%)
Diabetic cases without retinopathy	Mean ± S.D	2.03 ± 0.19	7.12 ± 0.94
Diabetic cases with retinopathy	Mean ± S.D	1.97 ± 0.35	8.39 ± 1.19
Diabetic cases without retinopathy vs. Diabetic cases with retinopathy	p-value	<0.05	<0.001

There was a substantial increase in mean HbA1c values in non-retinopathic and retinopathic diabetic patients relative to the control group ($p<0.001$), as shown in Tables 2, 3 & 4. Likewise, when contrasted with diabetic patients without retinopathy, there was a genuinely huge ascent in mean HbA1c levels in diabetic patients with retinopathy ($p<0.001$).
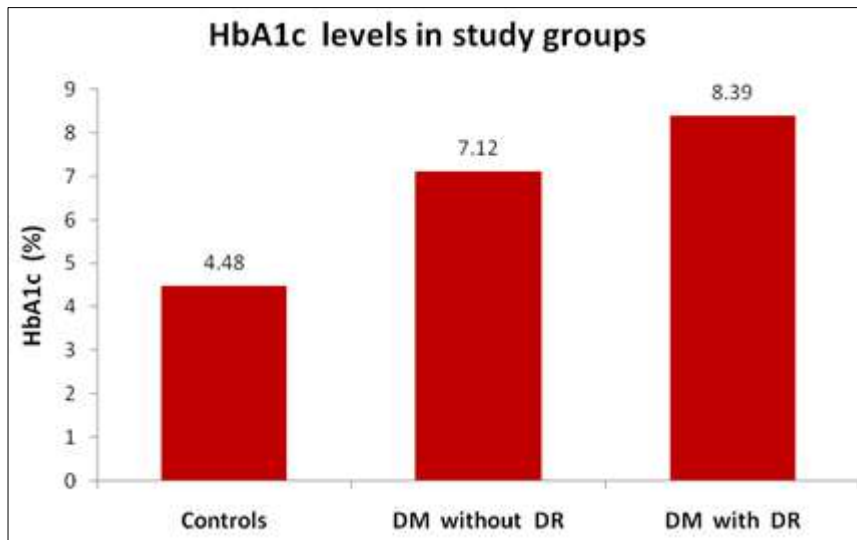

Fig 2: Bar diagram showing the mean HbA1c levels in the study groups

As appeared in Tables 2, 3 and 4, the mean magnesium levels in diabetic patients without retinopathy and with retinopathy were measurably essentially lower contrasted with the benchmark group ($p<0.001$). Also, in diabetic patients with retinopathy, there was a measurably huge drop in mean magnesium levels when contrasted with diabetic patients without retinopathy ($p<0.05$).
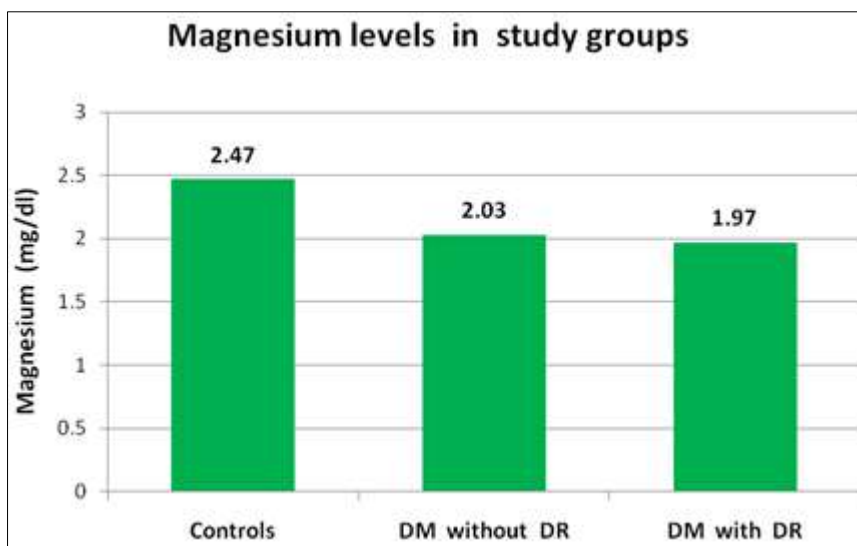

Fig 3: Bar diagram showing the mean serum magnesium levels in the study groups

The above bar graph demonstrates that comparative with diabetic patients without retinopathy and the benchmark groups, the mean magnesium esteems in the serum of diabetic patients with retinopathy are fundamentally lower.

Discussion

Diabetic retinopathy is one of the main sources of obtained visual deficiency and is a sight-threatening complication of diabetes mellitus. It is due to the retinal arterioles, capillaries, and venules being affected by microangiopathy. Both microvascular leakage and microvascular occlusion cause damage. The development and progression of retinopathy in diabetic patients have been linked to several risk factors.

The current research involved 120 participants, of which 80 were diabetic patients and 40 were generally healthy subjects. Out of 80 patients with diabetes, 40 were diabetic patients without retinopathy and 40 were diabetic patients with retinopathy.

The mean duration of diabetes was 3.64 ± 2.03 years in diabetic patients without retinopathy, and the mean duration of diabetes was 9.72 ± 3.64 years in diabetic patients with retinopathy. The mean duration of diabetes is more in diabetics with retinopathy when compared to diabetics without retinopathy, which is statistically significant (p-value < 0.001).

Our findings are equivalent with the investigation led by M Rema *et al.* who demonstrated that the diabetes mellitus period is potentially the best indicator of retinopathy advancement. Studies have additionally indicated that the danger of diabetic retinopathy increments by 1.89 occasions with each 5-year ascend in the length of diabetes mellitus [10]. Studies conducted by Farhan K H *et al.* have also shown that the duration of diabetes mellitus will increase the risk of diabetic retinopathy [11].

The mean HbA1c level in controls was 4.48 % and the mean HbA1c levels in diabetics without and with retinopathy were 7.12 % and 8.39%. There was an increase in HbA1c levels in diabetic groups when compared to controls which are statistically significant (p-value < 0.001). Within diabetic groups, HbA1c levels were more in diabetics with retinopathy when compared to diabetics without retinopathy, which is statistically significant (p-value < 0.001).

Expanded degrees of HbA1c in diabetic patients with retinopathy and diabetic patients without retinopathy have been noticed [6]. Studies have also shown that every 1 % rise in HbA1c in patients with type 2 diabetes mellitus would result in a 37 % increase in microvascular complications [12]. A significant ascent in the degrees of HbA1c in diabetic patients with retinopathy has appeared by KG Santos *et al.* [13] The consequences of this study and previous studies show that hyperglycemia is an intense pointer of the advancement of diabetic retinopathy, as appeared by the ascent in HbA1c levels. The potential cause is that hyperglycemia leads to nearly all protein glycation, resulting in the development of advanced end products of glycation. In a few tissues, including blood vessel dividers, these high level glycation final results cause cross-linkage of collagen and other extracellular lattice proteins [14]. Vascular injury caused by hyperglycemia leads to increased glucose flow through the polyol pathway, leading to cellular damage, resulting in numerous micro-vascular and macro-vascular complications [15].

HbA1c is also shown to have a special affinity for oxygen, thus causing tissue anoxia and playing a role in causing micro and macroangiopathy [6]. As mediators of microvascular permeability, ischemia & angiogenesis, the interaction of advanced glycation end products and their receptors has been involved [15].

Mean serum magnesium levels were 2.47 mg/dl in the controls and mean serum magnesium levels were 2.03 mg/dl and 1.97 mg/dl in diabetics without and with retinopathy. Compared to the control group, there was a statistically significant decrease in magnesium levels in the diabetic group (p-value < 0.001). Within the diabetic group, magnesium levels were more in diabetics with retinopathy when compared to diabetics without retinopathy, which is statistically significant (p-value < 0.05).

Our results agree with the study conducted by Hatwal *et al.*, Ishrat Kareem *et al.*, who showed that hypomagnesemia is a risk factor for retinopathy formation. The existence of hypomagnesemia in diabetic retinopathy has been shown [6, 17].

The exact reason for hypomagnesemia in unexplained instances of diabetes mellitus. The contributory factors may be low food intake, impaired magnesium absorption, increased urinary loss due to hyperglycemia, and osmotic diuresis. For several enzymes that play an important role in the metabolism of glucose, magnesium is essential. It is said that magnesium depletion reduces the sensitivity of insulin, thus raising the risk of secondary complications [18].

The monitoring of plasma magnesium levels in diabetic patients with complications is therefore critical. In these patients, magnesium supplementation may help to reduce the progression of retinopathy [19, 20].

The extent of hyperglycemia contributes to the development of advanced end products of glycation. The different microvascular problems result in these advanced glycation end products [15, 22]. Also, generalized microangiopathy may be due to retinopathy. K.G Santos *et al.* have shown that diabetes mellitus length, glycosylated hemoglobin, and albumin excretion concentrations are independently correlated with diabetic retinopathy [13]. Studies have shown that the cumulative effects of different risk factors are likely to be due to increased capillary permeability, microangiopathy, and retinal ischemia [21].

The beginning and advancement of retinopathy can be limited by early conclusions and ideal treatment in diabetic patients. Further studies of oral supplementation of magnesium in various stages of diabetic retinopathy may be important.

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