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HEALTH AND REHABILITATION

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EFFECT OF MAGNESIUM SUPPLEMENTATION ON REDUCTION OF HBA1C IN NORMOTENSIVE TYPE 2 DIABETIC PATIENTS TAKING ORAL ANTI-DIABETIC DRUGS

Original Research

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Acknowledgement: The authors gratefully acknowledge the support of the Department of Medicine, CMH Peshawar, and all participants who contributed to this study.

Conflict of Interest: None
Grant Support & Financial Support: None

ABSTRACT

Background: Type 2 diabetes mellitus is commonly accompanied by micronutrient imbalance, and magnesium deficiency has emerged as a potentially important contributor to poor glycemic control. Reduced magnesium levels may impair insulin secretion, worsen insulin resistance, and disrupt glucose metabolism. Although magnesium supplementation has shown potential benefit in improving glycemic indices, locally relevant evidence remains limited, particularly in normotensive patients with type 2 diabetes mellitus receiving oral anti-diabetic drugs without insulin or antihypertensive therapy.

Objective: To determine the effect of magnesium supplementation on reduction of HbA1c in normotensive patients with type 2 diabetes mellitus taking oral anti-diabetic drugs.

Methods: This comparative analytical study was conducted in the Department of Medicine, Combined Military Hospital, Peshawar, over six months. A total of 180 patients aged 30–65 years with known type 2 diabetes mellitus were enrolled through non-probability consecutive sampling after informed written consent. Only normotensive patients receiving oral anti-diabetic drugs were included. Participants were divided into two equal groups based on treatment status: one group received magnesium oxide 400 mg once daily in addition to routine oral anti-diabetic therapy, while the other continued standard oral anti-diabetic treatment alone. Demographic, clinical, and biochemical data were collected using a structured proforma. Variables included age, sex, duration of diabetes, body mass index, serum magnesium, fasting plasma glucose, and HbA1c. Data were analyzed using SPSS version 26. Independent-samples t-test and chi-square test were applied as appropriate, and $p<0.05$ was considered statistically significant.

Results: A total of 180 participants were analyzed, with 90 patients in each group. Baseline characteristics were comparable between the two groups. Mean age was 52.1 ± 7.8 years in the magnesium group and 51.4 ± 8.2 years in the control group. Baseline HbA1c was 8.61 ± 0.84% in the magnesium group and 8.58 ± 0.81% in the control group. After follow-up, HbA1c decreased to 7.99% in the magnesium group and 8.38% in the control group. Mean HbA1c reduction was significantly greater in the magnesium group (-0.62 ± 0.45%) than in the control group (-0.20 ± 0.40%), with a between-group mean difference of -0.42% (95% CI: -0.54 to -0.30; p=0.001). Hypomagnesemia was present in 34.4% of participants and was associated with significantly higher baseline HbA1c ($p<0.001$).

Conclusion: Magnesium supplementation was associated with better glycemic control and a greater reduction in HbA1c among normotensive patients with type 2 diabetes mellitus receiving oral anti-diabetic therapy. Assessment and correction of magnesium deficiency may offer a simple and affordable adjunctive strategy in diabetes management.

Keywords: Blood Glucose; Diabetes Mellitus, Type 2; Hemoglobin A, Glycosylated; Hypoglycemic Agents; Magnesium; Magnesium Oxide; Micronutrients

INTRODUCTION

Type 2 diabetes mellitus (T2DM) remains one of the most common and clinically important non-communicable diseases worldwide, placing a substantial burden on patients, healthcare systems, and national economies, particularly in low- and middle-income countries. It accounts for nearly 90–95% of all diabetes cases and is characterized by a complex interplay of insulin resistance and progressive pancreatic β-cell dysfunction, ultimately resulting in chronic hyperglycemia (1). The long-term consequences of uncontrolled glycemia are well recognized, including microvascular complications such as retinopathy, nephropathy, and neuropathy, as well as macrovascular events such as ischemic heart disease and stroke. Among the available indicators of glycemic status, glycated hemoglobin (HbA1c) is considered a reliable marker of long-term glucose control, and even modest reductions in HbA1c have been associated with meaningful reductions in the risk of diabetic complications (2). Despite major advances in pharmacological management and the widespread use of oral anti-diabetic drugs, a considerable proportion of patients with T2DM continue to experience suboptimal glycemic control (3). This persistent gap in treatment response has drawn attention toward factors beyond conventional drug therapy, including diet, lifestyle, medication adherence, disease duration, and progressive metabolic dysfunction. In this context, growing interest has emerged in the contribution of micronutrient disturbances to impaired glucose regulation. Among these, magnesium has received particular attention because of its central physiological role in glucose metabolism, insulin action, and cellular energy processes (4). Magnesium is the second most abundant intracellular cation and serves as a cofactor in more than 300 enzymatic reactions, many of which are directly involved in carbohydrate metabolism. It contributes to insulin secretion from pancreatic β-cells, facilitates insulin-receptor interaction, supports post-receptor signaling, and influences transmembrane glucose transport. In addition, magnesium has been implicated in the regulation of oxidative stress, inflammatory activity, and ionic balance, all of which are relevant to the pathophysiology of T2DM (5).

A growing body of evidence suggests that magnesium deficiency may worsen insulin resistance and impair insulin-mediated glucose uptake, thereby contributing to poor glycemic regulation (5). Hypomagnesemia has been reported frequently among individuals with T2DM, with prevalence estimates ranging from 13% to 48% across different populations (6). Several mechanisms may explain this association, including inadequate dietary intake, gastrointestinal losses, renal magnesium wasting, and increased urinary excretion secondary to osmotic diuresis. Importantly, the relationship between magnesium deficiency and diabetes appears to be bidirectional. On one hand, low magnesium levels may aggravate insulin resistance, glucose intolerance, and metabolic instability; on the other, chronic hyperglycemia itself may promote further magnesium depletion, creating a self-perpetuating cycle of worsening glycemic control (6,7). This reciprocal pattern offers a biologically plausible explanation for why some patients remain poorly controlled despite receiving standard oral anti-diabetic therapy. Within the past decade, randomized controlled trials and meta-analyses have evaluated whether oral magnesium supplementation can improve glycemic outcomes in patients with T2DM. Several studies have reported modest but statistically significant reductions in HbA1c and fasting plasma glucose, particularly among individuals with low baseline magnesium levels (8). However, the overall evidence remains inconsistent. Variations in study design, supplementation dose, magnesium formulation, treatment duration, baseline magnesium status, and patient selection have contributed to heterogeneity in reported findings. As a result, uncertainty persists regarding whether magnesium supplementation should be considered beneficial across all patients with T2DM or whether its value may be limited to specific clinical subgroups.

This uncertainty is especially relevant in the local and regional context, where evidence remains limited. Available Pakistani studies have largely focused on the prevalence of hypomagnesemia or have included heterogeneous diabetic populations, often involving hypertensive patients, insulin-treated individuals, or mixed treatment groups (9,10). Comparatively little attention has been given to normotensive patients with T2DM who are managed exclusively with oral anti-diabetic drugs, despite the fact that this represents a large and clinically relevant subgroup. Studying this population separately is important because it allows a clearer assessment of the independent contribution of magnesium supplementation to glycemic control, without the confounding influence of antihypertensive medications or insulin therapy. Given the high burden of T2DM, the frequent occurrence of magnesium deficiency, and the low cost and wide availability of magnesium supplements, the question of whether magnesium supplementation can serve as a useful adjunct to standard oral anti-diabetic treatment is of considerable practical importance. In view of this background, the present study was designed to address the question of whether magnesium supplementation leads to a reduction in HbA1c among normotensive patients with type 2 diabetes mellitus who are taking oral anti-diabetic drugs. It was hypothesized that adjunctive magnesium supplementation would improve long-term glycemic control in this population. Therefore, the objective of this study was to determine the effect of magnesium supplementation on the reduction of HbA1c in normotensive type 2 diabetic patients receiving oral anti-diabetic therapy, with the aim of generating context-specific evidence that may support more effective and affordable strategies for glycemic management in routine clinical practice (8-10).A

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METHODS

This comparative cross-sectional study was conducted in the Department of Medicine, Combined Military Hospital (CMH), Peshawar, over a period of six months, from June 2024 to December 2024. The study was designed to compare glycemic parameters, particularly glycated hemoglobin (HbA1c), among normotensive patients with type 2 diabetes mellitus who were receiving oral anti-diabetic drugs, with and without magnesium supplementation. A total of 180 patients were enrolled after obtaining informed written consent. The required sample size was calculated using the WHO sample size calculator at a 95% confidence level and 80% study power, based on the expected difference in HbA1c reported in previous literature. Participants were recruited through non-probability consecutive sampling from patients attending the study setting during the data collection period. Patients of either sex, aged 30 to 65 years, with previously diagnosed type 2 diabetes mellitus were considered eligible for inclusion. Only normotensive individuals were included, defined as those having blood pressure below 140/90 mmHg and not using any antihypertensive medication. In addition, eligible participants were required to have been taking oral anti-diabetic drugs for at least three months prior to enrollment. Patients were excluded if they were receiving insulin therapy, had a diagnosis of hypertension, impaired renal function indicated by serum creatinine greater than 1.5 mg/dL, chronic liver disease, heart failure, pregnancy or lactation, chronic diarrhea, malabsorption syndromes, or a history of current magnesium supplementation other than the defined study exposure group. These criteria were applied to reduce the influence of conditions that could independently alter serum magnesium levels or glycemic status and thereby confound the comparison.

For analytical purposes, the participants were divided into two groups on the basis of their existing treatment status at the time of assessment. One group consisted of patients who were taking magnesium oxide 400 mg orally once daily in addition to their regular oral anti-diabetic therapy, while the second group included patients receiving standard oral anti-diabetic drugs without magnesium supplementation. Data were collected at a single point in time using a structured study proforma designed for uniform documentation of demographic, clinical, and biochemical variables. The information recorded included age, sex, duration of diabetes, body mass index (BMI), medication history, fasting plasma glucose, serum magnesium level, and HbA1c. Blood pressure status and treatment history were also reviewed to confirm normotensive eligibility. BMI was determined using standard anthropometric assessment based on weight in kilograms and height in meters squared. Laboratory parameters were obtained through routine hospital-based biochemical assessment as per institutional practice. Data entry and statistical analysis were performed using Statistical Package for the Social Sciences (SPSS) version 26. Quantitative variables such as age, duration of diabetes, BMI, fasting plasma glucose, serum magnesium, and HbA1c were summarized as mean ± standard deviation, whereas categorical variables such as sex and treatment group were presented as frequencies and percentages. Comparison between the two groups was carried out using the independent-samples t-test for quantitative variables and the chi-square test for categorical variables, where appropriate. A p-value of less than 0.05 was taken as statistically significant. The findings were interpreted in light of the study objective to examine the association between magnesium supplementation and HbA1c levels among normotensive patients with type 2 diabetes mellitus receiving oral anti-diabetic treatment.

Ethical approval for the study was obtained from the Institutional Ethical Review Committee of CMH Peshawar prior to the commencement of data collection. Participation was entirely voluntary, and all enrolled patients provided informed written consent after being explained the purpose and nature of the study. Confidentiality and anonymity of patient information were maintained throughout the research process, and the collected data were used solely for academic and scientific purposes.

RESULTS

Data from 180 participants were analyzed, with 90 patients in the magnesium supplementation group and 90 in the control group. At baseline, the two groups were comparable across the measured demographic and clinical variables. The mean age was 52.1 ± 7.8 years in the magnesium group and 51.4 ± 8.2 years in the control group (p=0.56). Male participants constituted 60.0% (n=54) of the magnesium group and 57.8% (n=52) of the control group (p=0.76). The mean duration of type 2 diabetes mellitus was 6.8 ± 3.2 years in the magnesium group and 6.5 ± 3.1 years in the control group (p=0.58), while the mean body mass index was 28.4 ± 3.6 kg/m² and 28.1 ± 3.5 kg/m², respectively (p=0.62). Baseline glycemic and biochemical parameters were also similar between the groups, with mean HbA1c values of 8.61 ± 0.84% in the magnesium group and 8.58 ± 0.81% in the control group (p=0.82), and mean serum magnesium levels of 1.69 ± 0.21 mg/dL and 1.70 ± 0.20 mg/dL, respectively (p=0.78).

After three months, HbA1c levels decreased in both groups. In the magnesium supplementation group, the mean HbA1c decreased from 8.61 ± 0.84% to 7.99%, representing a mean reduction of 0.62 ± 0.45% (p<0.001). In the control group, the mean HbA1c decreased from 8.58 ± 0.81% to 8.38%, corresponding to a mean reduction of 0.20 ± 0.40% (p=0.002). The absolute difference in mean HbA1c between the two groups at three months was 0.39 percentage points, with a lower mean value observed in the magnesium group. When the mean change in HbA1c was compared between groups, the magnesium group demonstrated a larger reduction than the control group, with a between-group mean difference of -0.42% (95% CI: -0.54 to -0.30; p=0.001). Assessment of baseline serum magnesium status showed that 62 of the 180 participants (34.4%) had low serum magnesium levels, defined as less than 1.7 mg/dL, whereas 118 participants (65.6%) had normal magnesium levels of 1.7 mg/dL or above. The mean baseline HbA1c among patients with low serum

magnesium was 8.92 ± 0.79%, compared with 8.42 ± 0.78% among those with normal serum magnesium levels, and this difference was statistically significant (p<0.001).

Table 1. Baseline characteristics of participants (n = 180)

Variable	Magnesium Group (n=90)	Control Group (n=90)	p-value
Age (years), mean ± SD	52.1 ± 7.8	51.4 ± 8.2	0.56
Male gender, n (%)	54 (60.0)	52 (57.8)	0.76
Duration of T2DM (years), mean ± SD	6.8 ± 3.2	6.5 ± 3.1	0.58
BMI (kg/m²), mean ± SD	28.4 ± 3.6	28.1 ± 3.5	0.62
Baseline HbA1c (%), mean ± SD	8.61 ± 0.84	8.58 ± 0.81	0.82
Baseline serum Mg (mg/dL), mean ± SD	1.69 ± 0.21	1.70 ± 0.20	0.78

Table 2. Baseline serum magnesium status and HbA1c (n = 180)

Serum Magnesium Status	n (%)	Baseline HbA1c (%) mean ± SD	p-value
Low Mg (<1.7 mg/dL)	62 (34.4)	8.92 ± 0.79	<0.001
Normal Mg (≥1.7 mg/dL)	118 (65.6)	8.42 ± 0.78	

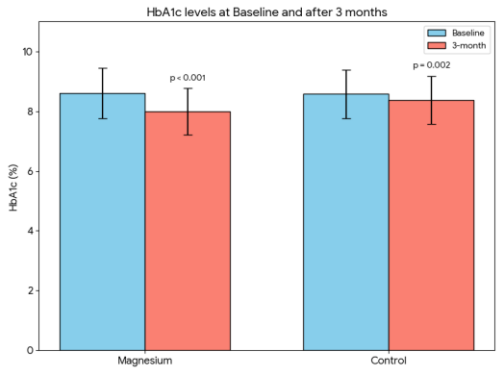

Figure 1: Comparison of Mean HbA1c Levels Between Magnesium and Control Groups

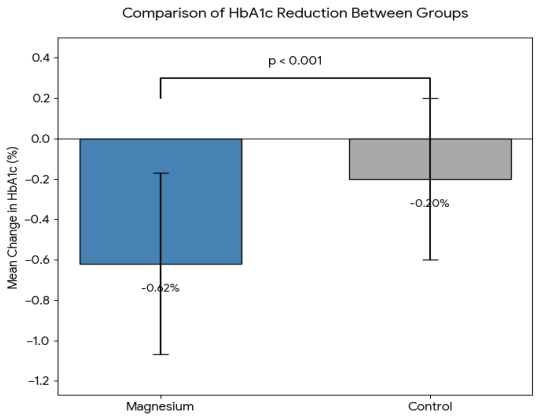

Figure.2. Comparison of Mean HbA1c Reduction (ΔHbA1c) Between Groups

DISCUSSION

The present study examined the association of magnesium supplementation with glycemic control in normotensive patients with type 2 diabetes mellitus who were receiving oral anti-diabetic therapy and found that patients using magnesium oxide 400 mg daily experienced a greater reduction in HbA1c over three months than those receiving standard oral therapy alone. Although improvement was observed in both groups during follow-up, the magnitude of HbA1c decline was more pronounced in the magnesium-supplemented group, suggesting an additional glycemic benefit when magnesium was used alongside routine oral anti-diabetic treatment (11). This finding was clinically relevant because even modest reductions in HbA1c are generally considered meaningful in the long-term reduction of diabetes-related complications, particularly when achieved through low-cost and readily available adjunctive measures. The observed findings were biologically plausible and were consistent with the known physiological role of magnesium in carbohydrate metabolism. Magnesium functions as an essential cofactor in numerous enzymatic reactions involved in glucose oxidation, ATP metabolism, insulin secretion, and post-receptor insulin signaling. It also contributes to transmembrane glucose transport and cellular insulin responsiveness. When magnesium status is suboptimal, these processes may become less efficient, thereby promoting insulin resistance and impaired glycemic regulation. In this context, correction of magnesium deficiency may reasonably contribute to improved insulin sensitivity and

better metabolic control, which may explain the greater decline in HbA1c observed in the supplemented group in the present study (12-14).

Another important finding was the association between low baseline serum magnesium and poorer baseline glycemic control. Patients with hypomagnesemia had higher baseline HbA1c values than those with normal serum magnesium levels, supporting the view that magnesium deficiency is not merely a biochemical abnormality in diabetes but may be linked to more disturbed glucose homeostasis. This observation was in agreement with the broader understanding that hypomagnesemia is common in type 2 diabetes mellitus and may result from multiple overlapping mechanisms, including reduced dietary intake, gastrointestinal losses, altered renal handling, and urinary magnesium wasting secondary to chronic hyperglycemia and osmotic diuresis. The coexistence of low magnesium and poor glycemic control further reflected the bidirectional relationship in which hyperglycemia promotes magnesium loss, while magnesium deficiency may in turn worsen insulin resistance and glucose intolerance, thereby reinforcing a vicious metabolic cycle (15,16). The results of the present study were broadly in line with previous trials, systematic reviews, and meta-analyses reporting modest but statistically significant improvements in HbA1c after oral magnesium supplementation in patients with type 2 diabetes mellitus. At the same time, the literature has remained somewhat heterogeneous, and the present findings should be interpreted within that wider context. Variability in baseline magnesium status, differences in the type and dose of magnesium salts, treatment duration, bioavailability, sample characteristics, and background anti-diabetic regimens have all contributed to inconsistency across studies. Magnesium oxide, which was used in the present study, is inexpensive and widely accessible, although it is generally considered less bioavailable than preparations such as magnesium citrate or magnesium chloride. Even so, the improvement observed with magnesium oxide in this study suggested that clinically useful benefit may still be achievable with this formulation, particularly in resource-constrained settings where cost and availability strongly influence therapeutic choices (17).

The local significance of the findings also deserved emphasis. Evidence from Pakistan on magnesium supplementation in diabetes has remained limited and has often involved mixed populations, including hypertensive patients, insulin-treated patients, or broadly defined diabetic cohorts. By focusing specifically on normotensive patients treated only with oral anti-diabetic drugs, the present study contributed more targeted evidence in a subgroup that is common in clinical practice but insufficiently studied in the local setting. This narrower clinical focus reduced some degree of heterogeneity and allowed a clearer view of the relationship between magnesium supplementation and HbA1c in patients without the confounding influence of antihypertensive therapy or insulin treatment. From a practical standpoint, the findings supported the possibility that magnesium supplementation may serve as a simple adjunctive strategy in selected patients whose glycemic control remains suboptimal despite oral therapy alone (18). At the same time, the findings required careful interpretation and did not justify overstatement. Although the supplemented group showed greater improvement, the study design and sampling approach limited the strength of causal inference. Non-probability consecutive sampling reduced the external generalizability of the findings, and the absence of random allocation increased the possibility of residual confounding. Factors such as dietary magnesium intake, medication adherence, physical activity, baseline disease severity, variation in oral anti-diabetic regimens, and lifestyle modifications during follow-up may all have influenced glycemic outcomes independently of supplementation. As a result, the findings were more appropriately interpreted as supportive of an association and potential adjunctive benefit rather than as definitive proof of causality. This distinction was particularly important because magnesium supplementation was evaluated in a real-world comparative context rather than under strictly controlled interventional conditions (19).

Several limitations therefore needed to be acknowledged. The follow-up duration of three months was sufficient to capture short-term change in HbA1c, but it did not permit assessment of long-term sustainability, diabetic complications, safety with prolonged use, or persistence of response after continued supplementation. Serum magnesium was used as the biochemical indicator of magnesium status, yet serum values represent only a small fraction of total body magnesium and may not fully reflect intracellular magnesium stores, which are more relevant to metabolic activity. The study also did not appear to account for dietary intake of magnesium-rich foods, gastrointestinal tolerance of supplementation, or changes in body weight and treatment adherence during follow-up. In addition, subgroup analysis according to baseline magnesium status would have been valuable, as patients with confirmed hypomagnesemia may derive greater benefit than those with normal levels. These methodological constraints did not negate the observed findings, but they did frame the degree of confidence with which the results could be applied to broader populations (19,20). Notwithstanding these limitations, the study had a number of strengths. It addressed a clinically relevant and underexplored question in a local setting, included a clearly defined patient population, and evaluated a low-cost intervention with direct practical implications. The comparison groups were similar in major baseline demographic and clinical characteristics, which strengthened the internal comparability of the analysis. The use of HbA1c as the primary outcome also enhanced the clinical value of the findings, as HbA1c remains one of the most meaningful markers of long-term glycemic control in routine diabetic care. Moreover, the inclusion of baseline serum magnesium data added depth to the analysis by linking biochemical status with glycemic burden rather than evaluating supplementation in isolation.

The implications of these findings were therefore cautiously encouraging. In routine clinical practice, assessment of serum magnesium may deserve greater attention in patients with type 2 diabetes mellitus, especially in those with persistently elevated HbA1c despite oral anti-diabetic treatment. Where low magnesium is identified, supplementation may represent a reasonable adjunct to standard management, particularly because magnesium preparations are inexpensive and widely available. In addition, nutritional counseling focused on magnesium-rich foods such as green leafy vegetables, legumes, nuts, and whole grains may complement pharmacological

strategies and provide a broader metabolic benefit. However, routine universal supplementation in all normotensive patients with type 2 diabetes mellitus would remain premature in the absence of stronger randomized evidence and clearer identification of the patient groups most likely to benefit. Future research should therefore move toward randomized controlled trials with longer follow-up periods, standardized magnesium formulations, careful monitoring of adherence, and more detailed assessment of dietary magnesium intake and concurrent therapies. Studies that stratify participants according to baseline hypomagnesemia, degree of glycemic derangement, duration of diabetes, and type of oral anti-diabetic regimen would be particularly valuable in identifying the most responsive subgroups. Additional work incorporating intracellular magnesium assessment, repeated biochemical monitoring, and clinical endpoints beyond HbA1c would also strengthen the evidence base. Taken together, the present study suggested that magnesium supplementation may have a useful adjunctive role in improving glycemic control among normotensive patients with type 2 diabetes mellitus receiving oral anti-diabetic drugs, while also underscoring the need for more rigorous and methodologically robust studies before broader recommendations can be made (20).

CONCLUSION

Magnesium supplementation appeared to support better glycemic control in normotensive patients with type 2 diabetes mellitus receiving oral anti-diabetic therapy, as reflected by a greater improvement in HbA1c compared with standard treatment alone. The study also highlighted that low serum magnesium was common and was associated with poorer baseline glycemic status, suggesting that magnesium deficiency may contribute to inadequate metabolic control in this population. These findings support the practical value of considering magnesium status during routine diabetic assessment and suggest that magnesium supplementation may serve as a simple, affordable, and accessible adjunct in selected patients, particularly those with persistent poor glycemic control. Overall, the study added clinically relevant local evidence and indicated the need for larger, well-designed studies to further clarify the long-term role of magnesium supplementation in diabetes management.

AUTHOR CONTRIBUTION

Author	Contribution
Dr Shah Nawaz Khan	Conceptualization, Methodology, Formal Analysis, Writing - Original Draft, Validation, Supervision
Dr Abid Sharif	Methodology, Investigation, Data Curation, Writing - Review & Editing
Dr Maryam Hussain	Investigation, Data Curation, Formal Analysis, Software
Dr Muhammad Yasin	Software, Validation, Writing - Original Draft
Dr Khayam	Formal Analysis, Writing - Review & Editing
Dr Syed Sibtain Maqsood	Writing - Review & Editing, Assistance with Data Curation

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