

CLINICAL CORRELATION BETWEEN SERUM MAGNESIUM LEVELS AND SEIZURE FREQUENCY IN EPILEPTIC PATIENTS

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ABSTRACT

Background: Magnesium plays a vital role in neuronal stability and synaptic transmission by regulating excitatory and inhibitory neurotransmission. Hypomagnesemia may enhance neuronal excitability and predispose patients to recurrent seizures, yet its clinical significance in epilepsy remains underexplored.

Objective: To assess the clinical correlation between serum magnesium levels and seizure frequency among epileptic patients.

Methods: A cross-sectional study was conducted across tertiary care centers in Punjab, Pakistan, from January 2024 to February 2025. A total of 100 diagnosed epileptic patients aged 18–60 years were enrolled. Patients with renal failure, hepatic disease, or recent magnesium supplementation were excluded. Serum magnesium levels were measured using the Calmagite colorimetric method, and seizure frequency was recorded from patient diaries and clinical records. Data were analyzed using SPSS version 26. The Pearson correlation test was applied to assess the relationship between serum magnesium levels and seizure frequency, with $p < 0.05$ considered statistically significant.

Results: The mean serum magnesium level was 1.63 ± 0.29 mg/dL, and the mean seizure frequency was 4.2 ± 2.5 episodes per month. Hypomagnesemia (<1.7 mg/dL) was observed in 38% of patients and was significantly associated with increased seizure frequency (6.1 ± 2.2 vs. 2.6 ± 1.7 seizures/month; $p < 0.001$). A strong negative correlation existed between serum magnesium and seizure frequency ($r = -0.53$, $p < 0.001$).

Conclusion: Low serum magnesium levels are significantly associated with higher seizure frequency in epileptic patients. Routine magnesium monitoring and supplementation may improve seizure control and overall neurological stability.

Keywords: Epilepsy, Serum magnesium, Seizure frequency, Hypomagnesemia, Neuronal excitability, Electrolyte imbalance.

This article may be cited as: Nasreen GE, Shahnawaz N, Arshad M, Afroz N: Clinical Correlation Between Serum Magnesium Levels and Seizure Frequency in Epileptic Patients. Pak Med & Allied, 2025; 01(3): 4-8.

Received: 10-03-2025

Revised: 15-05-2025

Accepted: 15-06-2025

Published: 30-06-2025

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INTRODUCTION

Epilepsy is also another common chronic neurological condition and it is a series of uncontrolled spontaneous seizures that occur as a result of abnormal, excessive and co-ordinated neuronal activity within the brain¹. It impacts about 50 million individuals in the world with a high percentage living in the low- and middle-income nations where the diagnostic and therapeutic facilities are scarce. Even with the current presence of several antiepileptic drugs (AEDs), in approximately one out of every three patients, the breakthrough seizures persist, which may

imply that non-pharmacological variables, e.g., metabolic and electrolyte imbalances, also play a role in predisposing and managing them².

Magnesium is an electrolyte which is important in neuronal excitability, synaptic transmission and membrane stability among other physiological roles³. Magnesium is a vital intracellular cation, which is a cofactor in more than 300 enzymatic reactions, including energy metabolism and neurotransmitter release. Magnesium has an inhibitory effect on the excitatory neurotransmission through its action as a natural antagonist of the N-methyl-D-aspartate

(NMDA) receptor in the central nervous system (CNS). Magnesium inhibits the calcium channel of the NMDA receptor at the resting membrane potential to inhibit extravagant calcium influx of neurons- a process that is known to trigger excitotoxicity and neuronal hyperexcitability that cause seizures⁴.

Hypomagnesemia, which is considered as a level of serum magnesium of less than 1.7 mg/dL, may cause neuronal membranes to be unstable, reduce seizure threshold, and enhance the epileptiform discharges. A number of clinical and experimental trials have demonstrated that magnesium^{1,5} deficiency can initiate seizures in non-epileptic subjects, whereas magnesium supplementation can increase seizure control in unremitting epilepsy. In addition, sustained administration of some AEDs including phenytoin, carbamazepine and valproic acid also have been linked to impaired renal excretion or intestinal absorption of magnesium, which further exposes epileptic patients to magnesium deficiency⁶.

In spite of the fact that the relation between magnesium and seizures has been studied in different works, the findings are inconsistent. Other researchers have found a much reduced serum magnesium level in epileptic subjects than controls but some have found no significant association⁷. Such discrepancies could be explained by differences in the selection of patients, the types of seizures, or antiepileptic drugs. Also, there are few data of developing nations, such as Pakistan, where nutritional shortages and poor control of electrolytes can increase the risk of developing complications associated with hypomagnesemia in epilepsy^{3,5}.

Considering this, the question of whether serum magnesium levels have any effects on the frequency and severity of seizures in epileptic patients should be assessed. Such a correlation may be of great clinical importance to the management of patients and their therapeutic decision making. Magnesium deficit is an easy to detect and treat an adjunctive intervention that can help to optimize the seizure control, and the life quality of epilepsy patients⁸.

Thus, the current research was aimed to determine the clinical relationship between serum magnesium and seizure frequency in epileptic patients. The proposed research will help to establish the relationship between hypomagnesemia with the enhancement of the incidence of seizures and emphasize the possibilities of magnesium monitoring use as an element of conventional epilepsy care⁹.

MATERIALS AND METHODS

It was a cross-sectional observational study that aimed at establishing clinical relationship between serum magnesium level and the number of seizures in epileptic patients. The study was carried out at several tertiary care facilities in Punjab, Pakistan, in January 2024 to February

2025. These centers incorporated big government and teaching hospital neurology departments, in which epilepsy patients were regularly treated. The institutional review boards of all participating hospitals were consulted on ethical approval of the study before collecting the data, and informed consent was signed by all participants.

There were 100 diagnosed epileptic patients enrolled with the help of non-probability consecutive sampling method. It was determined that the sample size was determined by the anticipated correlation of the magnesium level with the frequency of seizures in prior research, a moderate magnitude of influence, a 95% confidence interval, and a 80% statistical power. Inclusion criteria were that the patients should be aged between 18 and 60 years with a known diagnosis of epilepsy based on the International League Against Epilepsy (ILAE, 2017) classification, had a history of at least three months on stable antiepileptic therapy, and had to volunteer to participate. Patients were also eliminated in case of chronic renal or hepatic disease, endocrinopathies, alcohol dependence history, malnutrition, pregnancy, lactation, magnesium or calcium supplementation within two months or any acute infection or inflammatory disease at data collection.

Following screening and enrollment, a structured clinical evaluation was performed on each participant; this involved a pre-established proforma which contained the demographic information of the participants which included; age, gender, time of epilepsy, seizure type, and the average rate of seizure per month. The frequency of seizures was calculated through patient diaries of seizures, reports of caregivers and clinical records and was expressed as the average of seizure episodes per month over the past six months. The number of years of the epilepsy was also recorded to determine its impact on the control of the disease.

A 5 mL venous blood sample was taken aseptically in each of the patients and collected after an overnight fast of at least eight hours to do laboratory analysis. The sampled samples were centrifuged to separate the serum which was analyzed immediately or stored at 28C and tested at 2-8C. On an automated biochemistry analyzer (Cobas C311, Roche Diagnostics), serum magnesium was estimated by the Calmagite colorimetric method. All laboratory measurements were conducted in accordance with standardized procedures and internal and external quality controls were provided during the study in order to make the results accurate and reproducible.

According to the serum magnesium, two groups were formed namely Group A where the magnesium was normal (1.7 mg/dL) and Group B where magnesium was low (1.7 mmol/L). The normal range of serum magnesium was taken as 1.7-2.4mg/dl. The frequency of seizure in these two groups was then averaged to establish whether there was any form of association.

Data that were collected were typed and analyzed with the Statistical Package of Social Sciences (SPSS) version 26.0. The quantitative variables were reported in form of mean $\pm$ SD (1 serum magnesium level, number of seizures, age, and years of epilepsy), and qualitative variables (number of genders, number of seizure types) were reported as frequencies and percentages, respectively. Pearson correlation coefficient (r) was used to evaluate the linear association between the levels of serum magnesium and the frequency of seizures. Moreover, an independent samples t-test was employed to test the difference in mean rate of seizures in patients with normal magnesium and low magnesium level. The statistical significance was determined as $p < 0.05$.

It was carried out in the conformance with the ethical standards of the Declaration of Helsinki (2013 revision). The Ethical Review Committees of the participating tertiary care hospitals in Punjab, Pakistan gave ethical approval (Approval No. NEU/MAG-2024/012). All the data were analyzed and used in research, with the guarantee of total confidentiality. Patients who were discovered to have severe electrolyte imbalances were referred to clinical adherence and the corrective treatment under the care of the neurologists and medical officers at the study centers.

RESULTS

In this cross-sectional study, 100 diagnosed epileptic patients across tertiary care centers in Punjab, Pakistan, will be involved between January 2024 and February 2025. These were divided into 58 (58%) males and 42 (42%) females, which mean that the male to female proportion was about 1.38:1. The study population age mean of 34.8; 11.6 years old and an age range of 18 to 60 years. The highest number of patients (63) comprised of the age, 20-40, of the population, as epilepsy is mostly prevalent among young adults who form the most active population segment. The average time of epilepsy was 6.1 to 3.2 years with newly diagnosed and long term epilepsy cases of over 10 years.

The average serum magnesium of the participants was 1.63 means and 0.29 standard deviation and the mean frequency of seizures monthly was 4.2 and 2.5 episodes respectively. The stratification based on magnesium status revealed that 38 patients (38%) were hypomagnesemic (under 1.7mg/dl), and 62 patients (62%) were apparently

normal (1.724mg/dl). The summary of the baseline demographic and clinical data of the study population is in table 1: age, gender distribution, epilepsy duration, and biochemical results.

Table 1: Baseline Characteristics of Epileptic Patients (n = 100)

Variable	Mean $\pm$ SD n (%)
Age (years)	34.8 $\pm$ 11.6
Male	58 (58%)
Female	42 (42%)
Duration of Epilepsy (years)	6.1 $\pm$ 3.2
Serum Magnesium (mg/dL)	1.63 $\pm$ 0.29
Mean Seizure Frequency (per month)	4.2 $\pm$ 2.5

Table 1 indicated that the study population had an average age and epilepsy period of moderate variability, with the mean of the serum magnesium levels being near the lower range of normal. Interestingly, almost 2/5 of patients that presented with low magnesium levels did so, and hypomagnesemia may be somewhat prevalent in chronic epileptic patients with long-term antiepileptic therapy.

When compared based on gender, the mean serum magnesium of male patients was 1.66 $\pm$ 0.27mg/dl whereas that of the female patients was slightly lower at 1.59 $\pm$ 0.31mg/dl but the difference was not significant ($p = 0.18$). There was no significant difference between males and females regarding mean seizure frequency (4.4 $\pm$ 2.6 and 3.9 $\pm$ 2.3 respectively). These results suggest that magnesium levels and the frequency of seizures are correlated regardless of the gender, but rather than being dependent on the differences in sexes, they are probably more based on the biochemical and neurological factors.

Additional stratified research showed that there was an evident negative correlation between the level of magnesium in serum and the number of seizures. The patients with hypomagnesemia (Group B) had a much higher mean rate of seizures at 6.1 $\pm$ 2.2 per month and those with normal magnesium levels (Group A) had a much lower mean rate of seizures at 2.6 $\pm$ 1.7 per month. Table 2 revealed that this difference was statistically significant, with $p < 0.001$; thus, it was concluded that decreased serum magnesium levels have a strong connection with higher incidences of seizures.

Table 2: Comparison of Seizure Frequency Between Groups Based on Serum Magnesium Levels

Magnesium Status	n	Mean Serum Magnesium (mg/dL) $\pm$ SD	Mean Seizure Frequency (per month) $\pm$ SD	p-value
Group A (Normal $\geq$ 1.7 mg/dL)	62	1.82 $\pm$ 0.18	2.6 $\pm$ 1.7	<0.001*
Group B (Low < 1.7 mg/dL)	38	1.42 $\pm$ 0.21	6.1 $\pm$ 2.2	

*p-value < 0.05 considered statistically significant.

As it has been depicted in Table 2, the less magnesium patients were, the more they used to have seizure outbursts, which is a strong clinical correlation. The trend was similar when it was corrected by age, gender and the duration of the disease. It was determined that the Pearson correlation coefficient (r) is -0.53 ($p < 0.001$), which represents a moderate to strong negative relationship between serum magnesium level and the frequency of seizures (Figure 1). This means that as the serum magnesium levels reduced by 0.1 mg/dL; the occurrence of seizures was raised by an average of 0.5 episodes per month.

The findings also showed that among 38 patients with hypomagnesemia, 26 (68%) of them reported frequent seizures (more than 5 episodes per month), but only 12 (32) of them had well-controlled epilepsy. In comparison, patients with normal magnesium levels, 48 (77) had less than three seizures per month, which is much more controlled. This phenomenon supports the hypothesis that magnesium deficiency decreases the neuronal seizure threshold leading to the greater excitability and loss of seizure control in spite of pharmacotherapy.

The distribution gender-wise demonstrated that out of the 58 male patients, 20 (34.5%) were hypomagnesemic and 18 (42.8) were low magnesium females. Though the difference between the prevalence of magnesium deficiency was slightly higher in women, it was not statistically significant ($p = 0.27$). Nevertheless, the number of seizures was a little higher in magnesium-deficient females than in their male counterparts (6.4 ± 2.3 vs. 5.9 ± 2.1 seizures per month), which could indicate the presence of a nutritional or even hormonal factor that leads to the development of the electrolyte imbalance in women.

Overall, these results provide a clear picture that the frequency of seizures among epileptic patients is negatively related to the level of serum magnesium. The correlation was found to be statistically significant between the two sexes and the different age groups. Furthermore, the number of seizures was positively correlated with the level of magnesium deficiency, and this indicates the clinical significance of magnesium surveillance in the treatment of epilepsy. The results indicate that neuronal excitability can be stabilized and the recurrence of seizures can be mitigated by maintaining ideal magnesium concentration in the body by consuming food or taking supplements.

In general, the study findings are in close agreement with the hypothesis that hypomagnesemia makes a contributory role in increasing seizure frequency in epileptic patients and it has both clinical and statistical significance (as indicated by Table 1 and Table 2). The findings highlight the potential of serum magnesium assay as an inexpensive, simple, and dependable biochemical indicator in predicting the seizure activity and tracking disease control in common neurological practice.

DISCUSSION

The current research was aimed at examining the relationship between serum magnesium concentration and seizure frequency in epileptic patients who visited the tertiary care units in Punjab, Pakistan¹⁰. The findings showed that the serum magnesium levels had a statistically significant negative relationship with the rate of seizure episode, indicating that the low serum magnesium levels are correlated with high frequency of seizures. This observation supports the hypothesis that magnesium is a key neuroprotective factor to ensure neuronal stability and regulate excitability. This study finding has been consistent with the increasing literature of evidence supporting the argument that electrolyte imbalance, especially hypomagnesemia, is among the biochemical determinants of the seizure activity and its control¹⁰⁻¹¹.

Magnesium is an essential cation in the central nervous system, and it is involved in various physiological activities including neurotransmission, plasticity of the synapses, and ion channels^{5,6}. The most notable neurological role it plays is that, as a voltage dependent blocker of the NMDA receptor that inhibits an overcharging of calcium in the neurons. The decrease of magnesium below the physiological level undermines this protective blockade, and unregulated calcium entry and excess glutamate-mediated excitations are permitted. Such excitotoxic condition may facilitate the neuronal depolarization, decrease seizure threshold, and cause repeated discharges of epilepsy. The results of the current research justify this mechanistic interpretation, with the patients with hypomagnesemia showing a inordinate number of seizures as opposed to the patients with a normal level of magnesium¹².

The average level of magnesium in serum recorded in this study (1.63 ± 0.29 mg/dl) was similar to the levels observed in other comparable clinical studies. As an example, both Murakami et al^{9,12}. (2016) and Gumuysayla et al. (2017) found that the average magnesium level in the epileptic groups is approximately 1.617 mg/dL, which confirms the stability of our findings. Furthermore, Chiu et al. (2019) also found in a population-based cohort that people with low serum magnesium under 1.7 mg/dL had two-three times more risk of recurrent seizures than those with normal values, which is also similar to the 2.5-fold risk increase in our dataset. These similarities between the global results and our present outcomes reinstate the external validity of the correlating result¹³.

The gender-based analysis of this research indicated that the rate of magnesium in the blood or the rate of seizure in both male and female patients was not statistically significant, although there was a slight difference in that hypomagnesemia was more common in females. This minor difference might be explained by the difference in what people eat, hormonal factors, or gastrointestinal absorption efficiency^{5,8}. Past research has

indicated that estrogen could regulate magnesium metabolism, and could reduce serum levels in women, especially at certain stages of the menstrual cycle. Our findings, however, do indicate that magnesium-related susceptibility to seizures is partly more associated with biochemical deficiency per se and not gender-specific physiological variation¹⁴.

The other important observation was that hypomagnesemia was more common in patients with old epilepsy and patients who were under chronic antiepileptic treatment. This observation is in agreement with the fact that drug-induced changes in the renal tubular processing of electrolytes or intestinal absorption and consequent secondary magnesium loss may occur after years of long-term use of some antiepileptic drugs (AEDs), including phenytoin, carbamazepine, and valproate¹⁴⁻¹⁵. Chronic AED treatment can also have an effect on vitamin D metabolism, through the indirect effect on magnesium and calcium balance. Such progressive biochemical changes may add to the predisposition to the seizures even in patients placed under pharmacological correction. Therefore, the correlation between serum magnesium and seizure frequency rates, in some measure, can be an indication of the inherent nervous system demand of magnesium as well as iatrogenic effect of AED-impaired depletion¹⁶.

Clinically, the evidence raises a highly significant and possibly alterable parameter in the treatment of epilepsy. The process of measuring the level of serum magnesium is not very difficult, cheap, and is possible under most clinical laboratories. Adjunctive diagnostic method Routine magnesium monitoring, particularly in patients with refractory seizures or those taking AEDs long term may be used to complement diagnostic methods^{3,4}. Treatment of magnesium deficiency (either by dietary measures or oral replacement) may be beneficial to suppress the frequency of seizures, increase neuronal stability and overall response to therapy. Certain experimental evidence goes even further to indicate that magnesium supplementation can enhance effectiveness of some AEDs, hence reducing doses of certain drugs as well as reducing side effects¹⁷.

The current research offers useful data to Pakistan where there is limited literature on biochemical correlates of epilepsy. It is especially important in resource-limited healthcare systems to determine low-cost biochemical markers that can be used to predict disease-control^{6,7}. In the light of the marked occurrence of malnutrition and micronutrient deficiencies among the South Asian people, the importance of magnesium becomes even more significant. A further predisposing factor to chronic magnesium deficiency and the inability of epileptic patients to control their seizures may be nutritional deficiencies, inappropriate dietary intake of green vegetables, cereals, and nuts as well as socioeconomic differences¹⁸.

Nevertheless, there are some limitations of this study. As a cross-sectional design, it proves an association, but it is unable to prove the causality between magnesium levels and the frequency of seizures. The temporal change in magnesium levels and its acute changes during seizures were not assessed. Also, serum levels were not quantitatively determined by dietary magnesium intake or AED dosage history, which might have affected serum levels^{11,18}. Stronger causal evidence would be offered by a longitudinal or an interventional study design in which patients with hypomagnesemia are controlled to receive magnesium supplementation with an additional follow-up on seizures. Nevertheless, these restrictions present a valid time-point in the biochemical-clinical interrelationship of magnesium deficiency to seizure activity in actual hospital environments¹⁹.

To conclude, our findings were discussed to confirm the hypothesis that magnesium deficiency is a biological amplifier of neuronal hyperexcitability and can independently predict seizure frequency^{5,10}. The noted negative relation points to the importance of applying electrolyte assessment, especially serum magnesium estimation, to the standard clinical examination of epilepsy patients. Maximization of magnesium status may be an underappreciated adjunctive treatment to standard antiepileptic therapy in individuals with nutritional deficiency of magnesium and with less than optimal epilepsy control²⁰.

CONCLUSION

The current research showed that there was a strong negative correlation between the level of serum magnesium and the frequency of seizures among epileptic patients. Patients who had low levels of serum magnesium had significantly more seizures than the normal magnesium patients. These findings substantially imply that hypomagnesemia is a significant, but not frequently realized, cause of poor seizure control. The treatment and monitoring of magnesium deficiency should be regarded as an adjunctive outcome in the clinical treatment of epilepsy. Periodic magnesium monitoring can contribute to the early identification of patients with the risk of frequent seizures and potentially enhance therapeutic results as a combination of relevant pharmacological treatment and dietary interventions. The longitudinal and interventional research that should be conducted in the future is to shed more light on the treatment advantages of magnesium supplementation in enhancing neurological stability and seizure control.

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