Studies on Some Electrolytes in Patients with Myasthenia Gravis in Imo State Teaching Hospital, Orlu, Nigeria

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Abstract

Myasthenia gravis (MG) is a rare autoimmune neuromuscular disorder marked by fluctuating weakness of voluntary muscles. Although the disease primarily affects the neuromuscular junction, electrolyte imbalances can influence muscle function and may worsen symptoms. This study focused on evaluating serum sodium, potassium, and chloride levels in MG patients attending the neurology clinic at Imo State Teaching Hospital, Orlu. A cross-sectional study was conducted on 50 participants-25 MG patients and 25 healthy controls matched for age and sex. Five millliliters of blood samples were collected and analyzed using the ion-selective electrode method. The results showed that MG patients had significantly lower sodium (130.72 ± 3.84 mmol/L) and chloride (82.44 ± 7.59 mmol/L) levels compared with controls, while potassium levels (4.33 ± 0.53) mmol/L were significantly higher (p= 0.001 and < 0.0001). Potassium levels were significantly raised among ages 20-40 years compared to ages >40 years. No gender-related differences in electrolyte levels were found among MG patients. In conclusion, participants aged 20–40 years demonstrated higher potassium levels than those aged >40 years, likely due to better renal function, higher muscle mass, and dietary habits.These findings suggest that hyponatremia, hypochloremia, and mild hyperkalemia may be part of the biochemical profile in MG, emphasize the need for age-specific considerations in electrolyte monitoring and dietary counseling to maintain optimal potassium balance across the lifespan, and that monitoring these electrolytes could improve clinical management.

Key words: Sodium, Potassium, Chloride, Myasthenia Gravis

Introduction

Myasthenia gravis (MG) is a chronic, acquired autoimmune disorder caused by antibodies targeting components of the neuromuscular junction, most commonly the nicotinic acetylcholine receptor (nAChR) on the postsynaptic membrane. This immune-mediated attack reduces the efficiency of neuromuscular transmission, leading to

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skeletal muscle weakness that typically worsens with activity and improves with rest[1]. MG can affect any voluntary muscle group, but ocular, bulbar, and limb muscles are frequently involved[2].

Although the hallmark clinical problem in MG is neuromuscular weakness, systemic biochemical changes may influence disease expression. Electrolytes such as sodium, potassium, and chloride are critical for maintaining resting membrane potential, generating action potentials, and ensuring proper muscle contraction[3]. Even minor deviations from normal levels can significantly affect neuromuscular transmission and, in the context of MG, may exacerbate weakness or precipitate a myasthenic crisis[4].

Sodium plays an essential role in initiating and propagating action potentials. Hyponatremia can impair nerve conduction and muscle contractility, compounding the functional deficit already present in MG[5]. Chloride, although less frequently discussed, stabilizes the resting membrane potential and helps modulate muscle excitability. Hypochloremia can contribute to hyperexcitability and fatigue[6]. Potassium is vital for repolarization of muscle membranes, and both hypo- and hyperkalemia can cause muscle weakness[7]. Some MG medications, such as corticosteroids and diuretics, may inadvertently alter electrolyte levels[8]. For example, corticosteroids can cause sodium retention and potassium loss, whereas potassium-sparing agents or renal dysfunction may lead to hyperkalemia[9]. In addition, infections and dehydration common in MG crises can exacerbate electrolyte disturbances.

Despite the importance of electrolyte balance in neuromuscular physiology, limited studies have focused on sodium, potassium, and chloride levels in MG patients, particularly in African populations. This study therefore aimed to evaluate these electrolytes in MG patients compared with healthy controls, and to determine if gender influences electrolyte levels in this population.

Materials and Methods

Study Area

The study was conducted at Imo State Teaching Hospital (IMSUTH), Orlu, Nigeria, a tertiary healthcare facility serving Imo State and surrounding regions. The hospital has well-equipped neurology and laboratory medicine units.

Study Design

This was a cross-sectional comparative study involving two groups—MG patients and healthy controls.

Study Population

Fifty participants were recruited: 25 clinically diagnosed MG patients attending the neurology clinic and 25 apparently healthy, age - and sex-matched individuals without MG or other neurological disorders. Participants were aged between 2 and 75 years.

Ethical Consideration

Approval for this study was obtained from the Institutional Research Ethics Committee of IMSUTH(IMSUTH/CS/121/). All participants gave written informed consent before sample collection.

Method of Recruitment

Patients were recruited consecutively from the neurology clinic after confirming their diagnosis. Controls were recruited from hospital staff and the local community. Informed consent was obtained from all participants.

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Sample Collection

After an overnight fast, 5 ml of venous blood was drawn into serum separator tubes for electrolyte analysis. Samples were centrifuged at 300 rpm for 10 minutes to separate serum.

Laboratory Procedure

Electrolytes (sodium, potassium, and chloride) were measured using the Ion-Selective Electrode (ISE) method. This method detects the electrical potential generated by the specific ion activity in the serum, compared against calibration standards[10].

Statistical Analysis:

Data were analyzed using SPSS version 27. Results were expressed as mean ± standard deviation. Independent sample t-tests compared MG patients and controls. A p-value of <0.05 was considered statistically significant.

Results

Table1:MeanValuesof Sodium, Potassium and Chloride in Myasthenia gravis

Parameter	Test	Control	t-value	p-value
Sodium (mmol/l)	130.72±3.84	141.87±2.47	17.26	0.001*
Potassium (mmol/l)	4.33±0.53	3.90±0.31	4.97	<0.0001*
Chloride (mmol/l)	82.44±7.59	100.72±5.07	14.17	<0.0001*

Patients Versus Controls.

KEY:

***:** Significant p value

MG patients had significantly lower sodium (130.72 ± 3.84 mmol/L) and chloride (82.44 ± 7.59 mmol/L) levels than controls, and significantly higher potassium levels (4.33 ± 0.53 mmol/L) (p =0.001and < 0.0001). No significant differences were observed between male and female MG patients.

Table 2: Mean Values of Sodium, Potassium and Chloride in Male Versus Female Patients with Myasthenia gravis.

Parameter	Male	Female	t-value	p-value
Sodium (mmol/l)	130.72±3.89	130.72±3.86	0.01	0.978
Potassium (mmol/l)	4.31±0.54	4.36±0.53	0.35	0.732
Chloride (mmol/l)	82.20±7.78	82.68±7.54	0.22	0.826

Table 2 shows that there were no significant differences in the mean values of sodium(130.72±3.89)mmo/l, Potassium (4.31±0.54)mmol/l and Chlorides(82.20±7.78)mmol/l in male patients with Myasthenia gravis when compared to females (130.72±3.86)mmol/l, (4.36±0.53)mmol/l,(82.68±7.54)mmol/l respectively(t=0.01, p=0.978, t=0.35, p=0.732 and t=0.22, p=0.826).

Table 3: Comparison of the Mean Values of Sodium, Potassium and Chloride in 20-40years verses 40-70years Myasthenia gravis Patients

Parameter	(20-40)yrs	(>40)	t-value	p-value
Sodium (mmol/l)	129.94±3.83	131.12±3.84	1.03	0.308
Potassium (mmol/l)	4.61±0.62	4.19±0.42	2.86	0.006*
Chloride (mmol/l)	83.71±8.54	81.79±7.09	0.84	0.403

Key:

***:** Significant p value.

In table 3, potassium (4.61±0.62)mmol/l in patients with Myasthenia gravis of ages(20-40)years was significantly raised when compared to those of ages(>40)yrs (4.19±0.42)mmol/l (t=2.86, p=0.006).

There were no significant differences in the mean values of sodium(129.94±3.83)

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mmol/l and chlorides (83.71±8.54) mmol/l in patients with Myasthenia gravis of ages (20-40)years when compared to those of ages (>40)yrs (131.12±3.84)mmol/l and (81.79±7.09) mmol/l(t=1.03, p=0.308 and t=0.84, p=0.403).

Discussion

This study found that MG patients had hyponatremia, hypochloremia, and mild hyperkalemia compared to healthy controls. These findings suggest that electrolyte disturbances may be part of the metabolic profile in MG.

Our finding of lower sodium levels aligns with Diringer[3], who reported that chronic illness, medications, and altered renal sodium handling can lead to hyponatremia in neurological disorders. Sodium depletion can reduce the amplitude of action potentials, thereby worsening neuromuscular transmission deficits in MG.

The observed hypochloremia is consistent with the role of chloride in stabilizing the resting membrane potential. Fara et al.[4] also noted that low chloride can lead to increased muscle fatigue, especially in patients already predisposed to weakness. Chloride losses may be exacerbated by diuretic therapy or gastrointestinal disturbances.

Interestingly, we observed elevated potassium levels in MG patients, in contrast to studies by Qureshi et al.[11], who reported hypokalemia in MG patients on long-term corticosteroids. Our results may reflect differences in treatment regimens. Some patients in our setting may be on potassium-sparing medications, or have reduced renal clearance leading to potassium accumulation. Hyperkalemia, while less common in MG, can impair muscle membrane repolarization and contribute to weakness[7].

No significant gender differences in electrolyte levels were found, suggesting that these imbalances are more closely linked to disease pathology and treatment rather than sex-specific factors. This agrees with observations from Ozturk et al.[12], who reported that electrolyte alterations in MG were independent of sex but strongly related to disease severity and medication use.Discussion (Continuation)

The observed higher potassium levels among participants aged 20–40 years compared to those over 40 years could be explained by several physiological and lifestyle factors. In younger adults, renal function is generally more efficient, and the kidneys maintain potassium homeostasis more effectively, preventing excessive loss through urine. This efficient renal tubular reabsorption may contribute to slightly higher serum potassium concentrations in healthy younger individuals[13]. Additionally, younger adults are often more physically active, and increased muscle mass can lead to higher baseline intracellular potassium levels, since potassium is the predominant intracellular cation[14].

Dietary patterns could also play a role. Younger adults may consume more potassium-rich foods such as fruits, vegetables, and energy drinks, while older adults may have reduced dietary potassium intake due to appetite changes, chewing difficulties, or medical advice to limit potassium because of declining kidney function[15]. Furthermore, age-related decline in renal function even in apparently healthy individuals can result in a more conservative potassium balance in older age groups, with a tendency toward mild hypokalemia due to chronic diuretic use or gastrointestinal potassium losses[16].

Our findings are consistent with the report of Al-Dahhan et al.[17], who observed higher potassium levels in younger adults, attributing this to both dietary habits and muscle mass. Similarly, Musa et al.[18] reported that potassium levels tend to decrease gradually after the fourth decade of

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life, likely due to reduced glomerular filtration rate (GFR) and hormonal changes affecting electrolyte handling. In contrast, a study by Nielson et al.[19] did not find a significant difference in potassium levels between younger and older adults, suggesting that other variables such as comorbidities, medications, and sample size could influence the results.

Overall, while our results align with several previous studies, the age-related pattern of potassium levels may vary depending on population characteristics, health status, and lifestyle differences. These variations highlight the importance of considering demographic and clinical factors when interpreting electrolyte results in both research and clinical practice.

Given the role of electrolytes in nerve and muscle function, regular monitoring could help prevent symptom exacerbations and crises. Early detection of hyponatremia, hypochloremia, or hyperkalemia would allow timely correction, potentially improving patient quality of life.

Conclusion

Myasthenia gravis is associated with significant changes in serum electrolytes particularly reduced sodium and chloride, and increased potassium. These alterations may worsen muscle weakness and should be monitored as part of routine clinical care. Participants aged 20–40 years demonstrated higher potassium levels than those aged >40 years, likely due to better renal function, higher muscle mass, and dietary habits. These differences are largely physiological and align with findings from previous research. For individuals aged >40 years, lower potassium levels may be influenced by age-related renal decline, reduced muscle mass, and potential dietary insufficiency. These findings emphasize the need for age-specific considerations in electrolyte monitoring and dietary counseling to maintain optimal potassium balance across the lifespan. Addressing electrolyte imbalances may help optimize neuromuscular function and reduce the risk of crises in MG patients.

Conflicting Interest

There is no conflict of interest.

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