Section: General Medicine

Case Series

SERUM MAGNESIUM AS PROGNOSTIC INDICATOR IN OP COMPOUND POISONING

Ranjith V[1], S Gayathri[2], Anjana Wilfred S[3]

[1]Associate Professor, Department of General Medicine Mysore Medical College and Research Institute, Irwin Road, Mysore, India.
[2]Third Year Postgraduate Department of General Medicine, Mysore Medical College and Research Institute, Irwin Road, Mysore, India.
[3]Third year Postgraduate, Department of General Medicine, Mysore Medical College and Research Institute, Irwin Road, Mysore, India.

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Corresponding Author:
Dr. S Gayathri,
Third Year Postgraduate Department of General Medicine, Mysore Medical College and Research Institute, Irwin Road, Mysore, India.
Email: gayathris2198@gmail.com

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ABSTRACT

Background: Organophosphorus compounds are commonly used as pesticides not only in agriculture and horticulture but also in households for control of vector-borne diseases such as malaria, dengue, etc., and are responsible for about 2,00,000 deaths due to pesticide poisoning in the developing world. According to the WHO report 2012, worldwide, there were about 1,93,460 deaths due to unintentional poisoning and 3,70,000 deaths due to suicidal pesticide poisoning. They are widely used for selfharm. In Indian studies, the incidence of suicidal poisoning using OPC ranges from 10.3% to 43.8. In India, deaths due to OPC poisoning are more common in southern and central India. The mortality rate varies from 16.7% to 40% in the hospitalized patients. The currently available markers like plasma pseudocholinesterase have the following disadvantage; Normal cholinesterase level is based on population estimates and there is a wide distribution in the definition of normal. A person with a "high normal" level may become symptomatic with a "low normal" activity. Several individuals do not seem to possess a known baseline level. Hypomagnesemia in OP compound poisoning might be due to GI losses, vomiting, diarrhoea. Decreased Mg level at time of admission is correlated with poor outcome in the form of increased incidence of intermediate syndrome, mechanical ventilation and death.

Objectives of the Study: • To estimate the levels of serum magnesium levels in OP compound poisoning. • To correlate them with outcome.

Methods of Study: After taking the institutional ethical clearance for the study, purpose of study will be explained to the patient and attenders. Written informed consent will be taken from the subjects. Relevant history and clinical examination will be done.

Results and Interpretation: A total of 100 subjects admitted in KR Hospital Mysore with OPC poisoning were studied to know the prognostic efficacy of Serum Magnesium using POP score. Of the 100 study subjects, majority were males (84 %) and they belonged to age group of 21-40 years. Among the 100 subjects, majority of patient with poorer clinical profile as indicated by higher POP score had lower values of serum magnesium (Spearman rank -0.67). This study thus proves that the serum magnesium that is deranged in OP compound poisoning can serve as useful prognostic indicator in predicting outcome.

Conclusion: This study proves that Serum Magnesium is deranged in acute organophosphorus compound poisoning and that they can be used as an alternative to the conventional Serum Pseudocholinesterase in predicting outcomes for op compound poisoning.

Keywords: Organophosphate, Serum Magnesium, POP score, Plasma pseudocholine ester.

INTRODUCTION

OPCs are a diverse group of chemicals, including insecticides such as malathion, parathion, diazinon, fenthion, dichlorvos, and chlorpyrifos, as well as nerve gases like soman, sarin, tabun, and VX.[1] Serious poisonings due to the misuse of OP insecticides have been reported for more than four decades.[2] OPs are among the most widely used insecticides globally, sharing a common mechanism of action despite their diverse structures.[3] The toxicity of OP insecticides is primarily associated with the inhibition of acetylcholinesterase in nervous tissues, leading to severe neurological dysfunction.[4] Magnesium is a major intracellular cation crucial for neuromuscular activity, enzymatic function, and cellular energy metabolism.[5] Approximately 50% of total body magnesium resides in bone, while the majority of extra skeletal magnesium is intracellular.[6] Deficiencies in magnesium are associated with increased mortality and worse clinical outcomes, particularly in critical care settings.[7] Magnesium plays a vital role in conditions such as hypocalcemia, tetany, hypokalemia, arrhythmias, stroke, ischemic heart disease, and bronchial asthma(8). ACh is a neurotransmitter with widespread functionality in the nervous system.[9] ACh is present in parasympathetic and sympathetic ganglia, all postganglionic parasympathetic nerves, the postganglionic sympathetic nerve innervating sweat glands, and skeletal neuromuscular junctions.[10] Upon depolarization of an axon, ACh is released into the synaptic cleft, which activates the postsynaptic receptors, resulting in the propagation of an action potential.[11] Carboxylic ester hydrolases metabolize ACh into acetic acid and choline through hydrolysis. This process occurs rapidly, with choline being reabsorbed into the presynaptic nerve to be used for the synthesis of additional ACh.[12] The main enzymes responsible for this metabolism are AChE and BuChE.[13] AChE is located in nervous and skeletal tissues, as well as on erythrocyte membranes. BuChE is present in plasma and various organs such as the liver, heart, pancreas, and brain. However, the function of BuChE remains partially understood.[14]

The key feature of organophosphate insecticides is their capacity to inhibit carboxyl ester hydrolases, primarily focusing on AChE inhibition.[15] These insecticides inactivate AChE by phosphorylating the serine hydroxyl group on the enzyme. As AChE is essential in Ach degradation, its inhibition results in an accumulation of ACh within the synapse, resulting in excessive stimulation of both nicotinic and muscarinic receptors.[16] Overstimulation of nicotinic receptors at the neuromuscular junction can result in fasciculations and myoclonic jerking, eventually leading to flaccid paralysis due to depolarizing blocks.[17] Nicotinic receptors are also found in the adrenal glands, potentially causing symptoms such as hypertension, sweating, tachycardia, and leukocytosis with a left shift.[18] Organophosphate poisoning induces symptoms based on its action at muscarinic receptors. These effects typically develop more slowly than nicotinic receptor effects, occurring through a G-protein–coupled receptor mechanism. Muscarinic receptors can be located in both the parasympathetic and sympathetic nervous systems. The sympathetic nervous system leads to the overstimulation of sweat glands, resulting in excessive diaphoresis. The parasympathetic effects of organophosphate poisoning can manifest in various systems, affecting the heart, exocrine glands, and smooth muscles. Muscarinic overstimulation can cause severe life-threatening conditions such as bradycardia, bronchorrhea, and bronchospasm, which can lead to breathing difficulties.Excessive Ach in the CNS can cause CNS depression, leading to coma and seizures.

SERUM MAGNESIUM IN OP COMPOUND POISONING

Cholinergic Overstimulation and Muscle Function

Organophosphate (OP) compounds inhibit AChE, leading to accumulation of ACh at nerve synapses. This results in prolonged stimulation of muscles (fasciculations), glands, and other tissues.[19] Muscle overstimulation due to excessive acetylcholine release can increase the demand for magnesium. Magnesium is required for proper muscle relaxation, and disturbances in its homeostasis could lead to muscle cramps, spasms, or paralysis.[20]

Possible effect on serum magnesium: Increased muscle activity (such as spasms) may lead to a shift of magnesium into cells or tissues, which could theoretically lower serum magnesium levels[21]. However, this is more of a secondary effect rather than a direct consequence of the OPC itself.

Renal Dysfunction and Magnesium Imbalance

In severe cases of OP poisoning, rhabdomyolysis (muscle breakdown) may occur, resulting in the release of muscle contents like myoglobin into the bloodstream[22]. Myoglobin can cause AKI due to its nephrotoxic effects[23]. Renal dysfunction can impair the body's ability to properly regulate electrolyte levels, including magnesium. This may lead to hypermagnesemia (high serum magnesium), particularly if the kidneys are unable to excrete magnesium adequately.[24]

Possible effect on serum magnesium: In cases of renal impairment (such as from rhabdomyolysis), serum magnesium levels might be elevated due to impaired renal excretion.[24]

Magnesium and Neuromuscular Toxicity

OP poisoning can lead to neuromuscular toxicity, and the balance between calcium and magnesium is particularly important in muscle contraction and relaxation. Low magnesium levels could potentially exacerbate neuromuscular hyperexcitability, increasing the severity of symptoms like fasciculations, convulsions, and respiratory failure.

Magnesium competes with calcium at neuromuscular junctions and helps to stabilize the membrane potential. An imbalance could increase muscle excitability and worsen the effects of cholinergic stimulation from OP poisoning.

Summary of Effects on Serum Magnesium

- **Low serum magnesium:** Could result from increased muscle activity (spasms, fasciculations) or other shifts of magnesium into cells.[21]
- **High serum magnesium:** May occur due to renal dysfunction, particularly in cases where rhabdomyolysis has caused kidney damage and the body is unable to excrete magnesium properly.[24]

Clinical Relevance

1. **Monitoring Electrolytes:** In patients with suspected or confirmed OP poisoning, monitoring electrolytes, including serum magnesium, is important. Imbalances may worsen neuromuscular dysfunction and complicate the clinical picture.[20]
2. **Treatment Considerations:**

- If hypomagnesemia (low magnesium) is suspected, magnesium supplementation may be required to stabilize neuromuscular function.[25]
- If hypermagnesemia is present (due to renal impairment or other factors), treatment may involve adjusting magnesium levels through fluid management or medications that promote renal excretion, depending on kidney function.[24]

Conclusion

While serum magnesium is not commonly a focal point in OP compound poisoning, it can be affected indirectly by muscle activity and renal dysfunction.[26] Elevated magnesium levels could occur in the setting of rhabdomyolysis and kidney injury, while magnesium depletion could be associated with muscle hyperexcitability and increased neuromuscular toxicity.[25] Careful monitoring of serum magnesium, along with other electrolytes, is important in the management of OP poisoning.[26]

MATERIALS AND METHODS

Source of Data

- Admitted patients of K. R. HOSPITAL, Department of General Medicine, MMC&RI, MYSORE.

Methods of Data Collection

- **Study design:** Diagnostic/Correlative
- **Study period:** April 2023 to April 2024
- **Place of study:** K. R. HOSPITAL, MMC&RI, MYSORE
- **Sample size: 100**

Inclusion Criteria

- Patients who have consumed documented OPCs.
- Patients who are more than 18 years old.

Exclusion Criteria

- Patients with mixed poisoning have been excluded.
- Patients with known medical illness such as chronic liver diseases, pancreatitis, malignancy, myopathy, renal failure, autoimmune diseases have been excluded.
- Pregnant patients were excluded.
- Patients who were on chronic drug usage with statins, steroids, diuretics

Methodology

- After obtaining approval and ethical clearance from the institutional ethics committee, the patients fulfilling the inclusion criteria and exclusion criteria will be enrolled for the study.
- After explaining the study to the patient, informed written consent will be obtained.
- Detailed history will be taken,detailed relevant clinical examination will be done and entered in a pre-structured proforma The following investigation will be done in this study
- Serum Magnesium

Assessment tools:

- Pre structured proforma.

Outcome measures

Efficacy parameters (Clinical outcome parameters).
POP Score.

Statistical Analysis

Data obtained from the study has been entered in the excel sheets and analyzed using SPSS software version 22.0 and will be presented as descriptive statistics in the form of frequency, tables, figures and graphs.

RESULTS

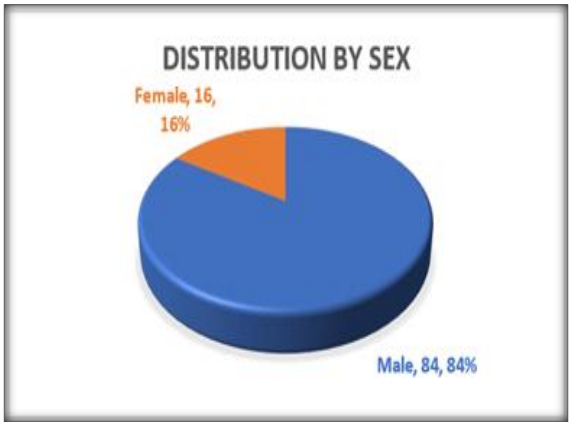

Figure 1: Distribution of subjects based on gender

Table presents the gender distribution of 100 subjects, with 84 % identified as males and 16% identified as females.

Table 1: Distribution of subjects based on gender

	N		Minimum		Maximum		Mean		SD
Male		84		21		70		43	12.73513
Female		16		18		63		38	12.52714
Total		100		18		70		42	12.73513

Table presents the age distribution of 100 subjects, ranging from 18 years to 70 years with mean age of 42 years and a standard deviation of 1.2 years.

Table 2: Mean age distribution of subjects

Age Group	No. of Subjects		Percentage
<30 Years		24	24%
31 - 40 Years		19	19%
41 - 50 Years		31	31%
51 - 60 Years		19	19%
> 60 Years		7	7%
Total		100	100%

Table illustrates the distribution of 100 subjects across various age groups. The largest age group was 41-50 years comprising 31% of sample, followed by people of age group less than 30 years. Those aged between 31-40 years and 51-60 years constituted 19% respectively and least represented group was people above 60 years of age.

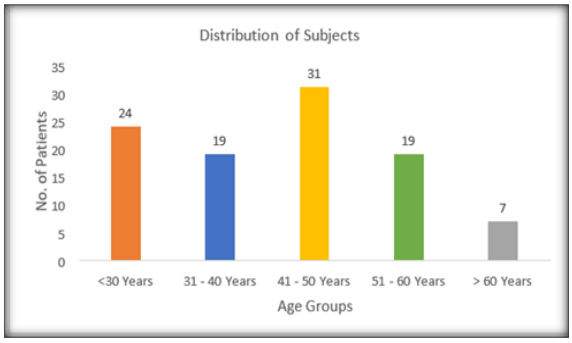

Figure 1: Distribution of subjects based on age groups

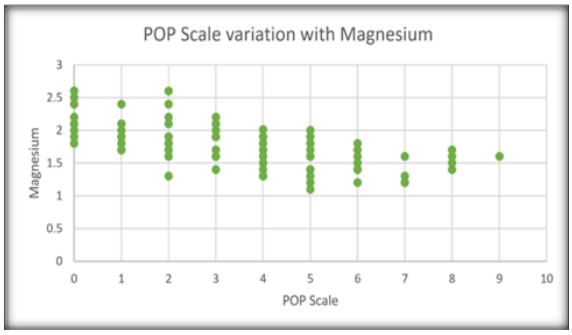

Figure 2: Scatter plot showing correlation between serum magnesium and POP score

This scatter plot shows co relation between POP score (x axis) and Serum Magnesium (y axis). The downward pattern of the plot indicates that there is a negative correlation between serum magnesium values and POP score.

Spearman's rank for the above-mentioned plot is -0.67 indicating negative correlation between serum magnesium values and higher values of POP score.

DISCUSSION

This study was conducted to see the usefulness of Serum Magnesium as prognostic indicators in OP compound poisoning comparing it with POP score.

In this study we found out that there is negative correlation between Serum Magnesium to higher values of POP score. In this study we found that majority of patients were between age groups of 20-40 and incidence of cases was higher among men which is similar to the study done by T. Selvaraj, which found out that a significant number of patients poisoned by OP compound were aged between 21-40 years. In our study, we found that subjects with initial low values of Serum Magnesium had a poorer prognosis which co related with study done by Sudhakar et al. at Kurnool medical College, Andhra Pradesh which reveals that it could be used as a biomarker in acute poisoning with OP compound. OP poisoning is a major public health issue in India, especially due to widespread pesticide use in agriculture. It leads to severe neurological symptoms by inhibiting acetylcholinesterase. India reports a high incidence of both accidental and intentional OP poisoning cases. Early and accurate diagnosis is critical but challenging with current clinical methods. Hence, there's a growing need for reliable and cheap biomarkers like the ones tested in our study to improve detection and treatment outcomes.

CONCLUSION

This study proves that the biochemical marker like Serum Magnesium is deranged in acute OPC poisoning and that they can be used as an alternative to the conventional Serum Pseudocholinesterase in predicting outcomes for OPC poisoning.

Summary

A total of 100 subjects admitted in KR Hospital Mysore with OPC poisoning were studied to know the prognostic efficacy of Serum Magnesium using POP score.

Of the 100 study subjects, majority were males (84 %) and they belonged to age group of 21-40 years.

Among the 100 subjects, majority of patient with poorer clinical profile as indicated by higher POP

score had lower values of serum magnesium (Spearman rank -0.67).

This study thus proves that the various biomarkers that are deranged in OP compound poisoning can serve as useful prognostic indicators in predicting outcome.

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