

ORIGINAL RESEARCH

Effectiveness of serum glucose/potassium ratio as a tool for predictor of intermediate syndrome

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ABSTRACT

Introduction: Intermediate Syndrome (IMS) is a delayed-onset neuromuscular complication that develops 24–96 hours after acute organophosphate (OP) poisoning. It is marked by proximal muscle weakness and respiratory distress, significantly increasing the risk of morbidity and mortality if not recognized early. Although supportive care is the cornerstone of management, the underutilization of reliable early biomarkers hampers timely diagnosis and intervention, leading to poorer clinical outcomes in affected patients. **Aim and Objective:** This study aims to evaluate the effectiveness of the serum glucose-to-potassium (GLU/K) ratio as a predictive biomarker for IMS in patients with OP poisoning, thereby facilitating earlier diagnosis and intervention. **Materials & Methods:** A 12-month prospective observational study was conducted at Shri B M Patil Medical College, Vijayapura, involving 228 patients over 18 years with confirmed organophosphate (OP) poisoning. Patients with age under 18, pregnancy, diabetes mellitus, renal disorders, or non-OP poisoning were excluded. Key biochemical parameters including serum glucose, potassium, and the glucose/potassium (GLU/K) ratio were measured, and patients were closely monitored for clinical progression to Intermediate Syndrome (IMS). **Results:** Of the 228 patients, 21 (9.2%) developed IMS. All IMS patients exhibited significant hypokalemia (mean $K^+ = 2.8$ mmol/L) and hyperglycemia (mean glucose = 226.4 mg/dL). The GLU/K ratio was significantly elevated in IMS patients (mean = 82.5) compared to non-IMS (mean = 40.5) with a p-value < 0.0001. ROC analysis showed an AUC of 0.910, with 94.3% sensitivity and 93.1% specificity. **Conclusion:** The GLU/K ratio is a reliable, accessible predictor of IMS, enabling early identification of high-risk patients. Early recognition through this biomarker can guide timely supportive interventions, especially in resource-limited settings.

Keywords: Organophosphate poisoning; Intermediate Syndrome; GLU/K ratio; hypokalemia; biomarkers;

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INTRODUCTION

Intermediate Syndrome (IMS) is a serious, delayed-onset neuromuscular complication that follows acute organophosphate (OP) poisoning. Organophosphates, commonly found in agricultural pesticides and insecticides, function by inhibiting acetylcholinesterase, the enzyme responsible for breaking down acetylcholine at neuromuscular junctions. The resulting accumulation of acetylcholine leads to overstimulation of cholinergic receptors, producing toxic effects that unfold in three main phases: an acute cholinergic crisis, followed by IMS, and finally delayed polyneuropathy. IMS typically emerges 24 to 96 hours after the resolution of the acute cholinergic phase (1). This syndrome is marked by symmetric muscle weakness, especially in the

proximal limbs, neck flexors, and respiratory muscles, significantly increasing the risk of respiratory failure. Unlike the initial cholinergic crisis—characterized by symptoms like salivation, lacrimation, urination, defecation, and bronchospasm—IMS is less responsive to antidotes like atropine and oximes, indicating a secondary and more resistant form of toxicity (2).

The pathophysiology of IMS remains incompletely understood, but it is believed to be related to neuromuscular junction dysfunction, potentially caused by the lingering effects of OP compounds or their metabolites. Clinical manifestations include symmetrical muscle weakness beginning in proximal limb muscles and neck flexors, which may progress to affect respiratory function. Bulbar palsy may also

occur, resulting in difficulty swallowing and speaking. Importantly, cranial nerve function is typically spared, helping to distinguish IMS from other neuromuscular conditions. Diagnosis of IMS is primarily clinical, relying on the history of OP exposure, timing of symptom onset, and the typical muscle involvement pattern. Nerve conduction studies may reveal reduced compound muscle action potentials, suggesting impaired neuromuscular transmission (3).

Management of Intermediate Syndrome (IMS) focuses on supportive care, particularly respiratory support, as atropine and oximes are less effective in this phase. Most patients recover fully, though recovery can take days to weeks. Early recognition is critical to prevent respiratory failure. IMS severity ranges from mild to severe. Mild cases involve weakness in neck and shoulder muscles without respiratory issues and typically respond well to monitoring. Moderate cases show trunk muscle weakness and early respiratory distress, sometimes requiring ventilation. Severe IMS includes profound respiratory muscle paralysis, often needing prolonged mechanical support and may lead to complications like aspiration and pneumonia (4).

Another way to categorize IMS is based on timing, distinguishing early-onset (within 24–48 hours) from late-onset (up to 96 hours post-exposure). Late-onset IMS is more unpredictable and may require extended supportive care. Multiple factors influence the development of IMS. Central among them is the prolonged inhibition of acetylcholinesterase, which leads to sustained acetylcholine accumulation and neuromuscular overstimulation (5). The type and dose of the organophosphate compound play critical roles, as some OPs form more stable and irreversible bonds with acetylcholinesterase, making treatment more difficult. High doses increase the likelihood of prolonged enzyme inhibition and severe toxicity. Delay in treatment also significantly raises the risk of IMS. Early and aggressive administration of antidotes can prevent neuromuscular dysfunction, but if intervention is delayed, residual neuromuscular damage may result in IMS (6).

Patient-specific factors such as advanced age, malnutrition, and underlying health conditions—especially liver or kidney dysfunction—can impair the clearance of organophosphates, increasing susceptibility to neuromuscular damage. Proximal and respiratory muscles are particularly sensitive to cholinergic overstimulation, explaining the typical weakness seen in Intermediate Syndrome (IMS). Early prediction of IMS is essential and relies on both clinical and biochemical indicators. Severe symptoms during the acute cholinergic phase—like seizures, unconsciousness, or need for mechanical ventilation—suggest significant acetylcholinesterase inhibition and a higher risk of IMS (7). Lipophilic organophosphates, which persist longer in fat tissues, also elevate risk. Delayed treatment further exacerbates toxicity. Biochemical markers, such as

persistently low acetylcholinesterase activity in plasma and red blood cells, signal ongoing enzyme inhibition and increased IMS risk. Additionally, pre-existing respiratory or neuromuscular disorders may heighten vulnerability. A prolonged or complicated recovery from the acute phase can also serve as a warning sign, indicating more severe or persistent toxic effects likely to progress to IMS (8).

Recently, the serum glucose/potassium (G/K) ratio has emerged as a promising early biomarker for predicting IMS. Organophosphate poisoning disrupts metabolic and endocrine systems by triggering a sympathetic response, leading to hyperglycemia through increased gluconeogenesis and glycogenolysis. Simultaneously, hypokalemia occurs due to acetylcholine-induced insulin release that shifts potassium into cells. Together, these metabolic effects elevate the G/K ratio. Studies have found that a high G/K ratio correlates with increased risk for IMS, likely reflecting the severity of cholinergic overstimulation and metabolic stress. The ratio is simple to calculate using routine blood tests, making it a practical tool in emergency settings (9).

An elevated glucose/potassium (G/K) ratio serves as an early warning indicator for identifying patients at high risk of developing Intermediate Syndrome (IMS) after organophosphate poisoning. It facilitates timely interventions such as respiratory support and aids in predicting disease severity and recovery duration (10). This is especially valuable in resource-limited settings or large-scale poisoning events. However, the G/K ratio's accuracy may be affected by underlying conditions like diabetes or renal disease, as well as by treatment timing. Therefore, it should be used in combination with clinical signs and other laboratory findings for a more accurate and comprehensive assessment (11).

This study aims to evaluate the serum glucose/potassium ratio as a predictive tool for identifying intermediate syndrome in cases of organophosphate poisoning, with the objective of enabling earlier diagnosis and improved clinical management through a simple, accessible biochemical marker.

MATERIALS AND METHODS

This prospective observational study was conducted over 12 months at Shri B M Patil Medical College, Vijayapura, to evaluate the serum glucose/potassium (GLU/K) ratio as a predictor of intermediate syndrome in organophosphate (OP) poisoning. Patients aged over 18 with confirmed OP poisoning were included. Exclusion criteria were age below 18, pregnancy, diabetes mellitus, chronic or acute kidney disease, and poisoning from non-OP substances. Ethical clearance was obtained, and informed consent was taken. Clinical evaluation and poison detection tests were performed, and biochemical parameters were analyzed to assess the association between

GLU/K ratio and the development of intermediate syndrome.

RESULTS

Table 1: Demographic features

Demographic Characteristics	Total (n=228)	No Intermediate Syndrome (n=207)	Intermediate Syndrome (n=21)
Sex			
Female	99 (43.2%)	89 (39.0%)	10 (4.4%)
Male	129 (56.8%)	118 (51.7%)	11 (4.8%)
Age (years)			
>18<25	112 (49.0%)	104(45.6%)	8 (3.5%)
25<35	54 (23.9%)	51 (22.3%)	3 (1.3%)
35<45	28 (12.3%)	23 (10.0%)	5(2.1%)
45<55	16 (7.0%)	13 (5.8%)	3 (1.2%)
>55	18 (7.8%)	16 (7.0%)	2 (0.87%)
Occupation			
Farmer	33 (14.4%)	27 (11.8%)	6 (2.6%)
Civil employee	23 (10.3%)	21(9.2%)	2 (0.87%)
Student	7 (2.9%)	4 (1.6%)	3 (1.2%)
Manual worker	77 (33.7%)	72 (31.5%)	5 (2.1%)
Housewives	47 (20.6%)	46 (20.1%)	1 (0.43 %)
Unemployed	32 (14.0%)	28 (12.2%)	4(1.7%)
Residence			
Rural	161 (70.8%)	145 (63.5%)	16 (7.0%)
Urban	67 (29.2%)	62 (27.2%)	5 (2.2%)

Occupation appears significantly associated with IMS development, whereas sex, age, and residence show no notable correlation. These results highlight occupation as a key demographic predictor, offering

valuable insights for clinical evaluation and management. Understanding such associations can enhance early identification and targeted intervention for individuals at risk of IMS.

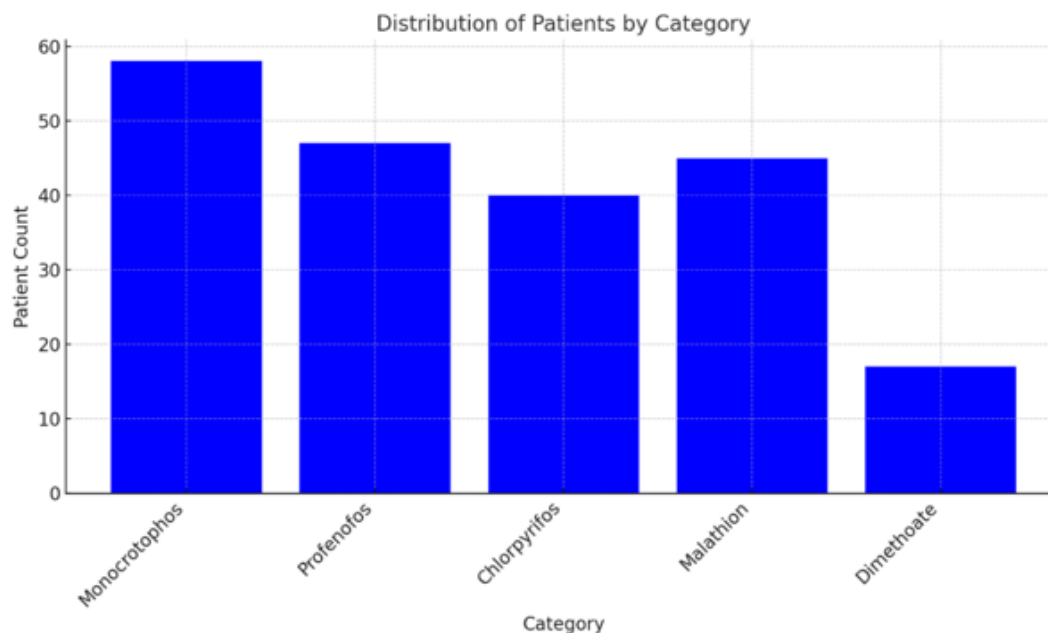


Figure1: Distribution of patients diagnosed with acute Organophosphorus Compounds OPCs exposure (Intermediate Syndrome IMS (-)) according to the type of the involved substance

Among patients without IMS, Monocrotophos is the most common toxic substance, followed by Profenofos and Malathion, while Dimethoate accounts for the fewest cases. This distribution underscores the need for targeted clinical focus on predominant agents, while also emphasizing the importance of broad awareness due to varied toxic exposures.

Table 2: By substance type, the percentage and distribution of individuals with acute exposure to Organophosphorus Compounds (OPCs) (Intermediate Syndrome IMS (+))

Category	Patient Count	Percentage
monocrotophos	3	14.2
Profenofos	2	9.5
Chlorpyrifos	3	14.2
(Lice powder) malathion	6	28.5
Dimethoate	7	33.3

Among 21 patients with acute substance exposure, Dimethoate is the most frequently encountered toxin, indicating the need for focused preventive and management strategies. The range of substances

involved reflects the complexity of toxic exposures, reinforcing the necessity for a broad-spectrum approach in clinical diagnosis and treatment planning.

Table 3: Comparison between patients diagnosed with intermediate syndrome IMS and those without intermediate syndrome IMS

Category	IMS (n=207)	%	No IMS (n=21)	%	Test of significance	P value
monocrotophos	58	28.0	3	14.2	χ^2 14.97	0.0048
Profenofos	47	22.7	2	9.5		
Chlorpyrifos	40	19.3	3	14.2		
malathion	45	21.7	6	28.5		
Dimethoate	17	8.2	7	33.3		

A χ^2 value of 14.97 and p-value of 0.0048 indicate a significant difference between the observed and expected distributions in IMS and No IMS groups. This suggests that specific toxic agents, such as Monocrotophos and Chlorpyrifos, may be more strongly associated with IMS development compared to other substances.

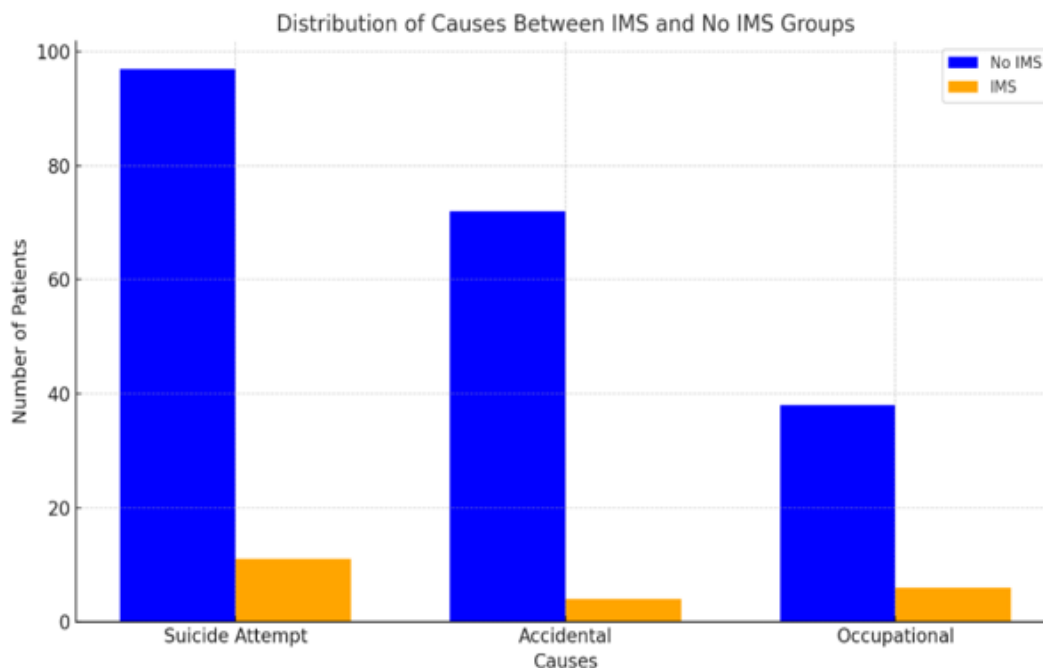


Figure 2: Distribution of causes between IMS and without IMS group

The distribution of exposure causes—Suicide Attempt, Accidental, and Occupational—shows higher patient numbers in the No IMS group across all categories. Although Suicide Attempt is the most common cause, the Chi-squared test ($p=0.277$) indicates no significant association between cause of exposure and IMS development, suggesting no disproportionate linkage.

Table 4: Comparison between patients diagnosed with intermediate syndrome IMS and those without intermediate syndrome IMS related to the main presenting Laboratory complaints

Laboratory investigations	Total (n=228)	Primary outcome		P value
		No intermediate syndrome(n=207)	Intermediate syndrome(n=21)	
Random blood glucose level(mg/dl)				< 0.0001
Mean±SD.	120 ±58.1	122±33.2	226.4±80.2	
Min.–Max	59.0–490.0	59.0–450.0	93.0–490.0	
Na(mmol\L)				1.0
Median	138.5 (137–144.8)	140.0(136.7–144)	152.0(137.5145.7)	
K(mmol\L)				< 0.0001
Mean±SD	3.5±0.7	3.7±0.5	2.8±0.6	
Min.–Max	1.9–5.5	2.6–5.5	72.9±29.8	

GLU/Kratio (mmol/L)				< 0.0001
Mean±SD	40.5±23.8	24.7±12.6	82.5±29.8	
Min.–Max	15.9–173.0	15.9–173.0	26.9–167.5	

Laboratory results from 228 patients—21 with IMS and 207 without—include Random Blood Glucose, Sodium (Na), Potassium (K), and the GLU/K ratio. Significant differences were observed in Blood Glucose, Potassium, and GLU/K ratio, suggesting their potential role in predicting IMS. Sodium levels showed no significant difference between groups.

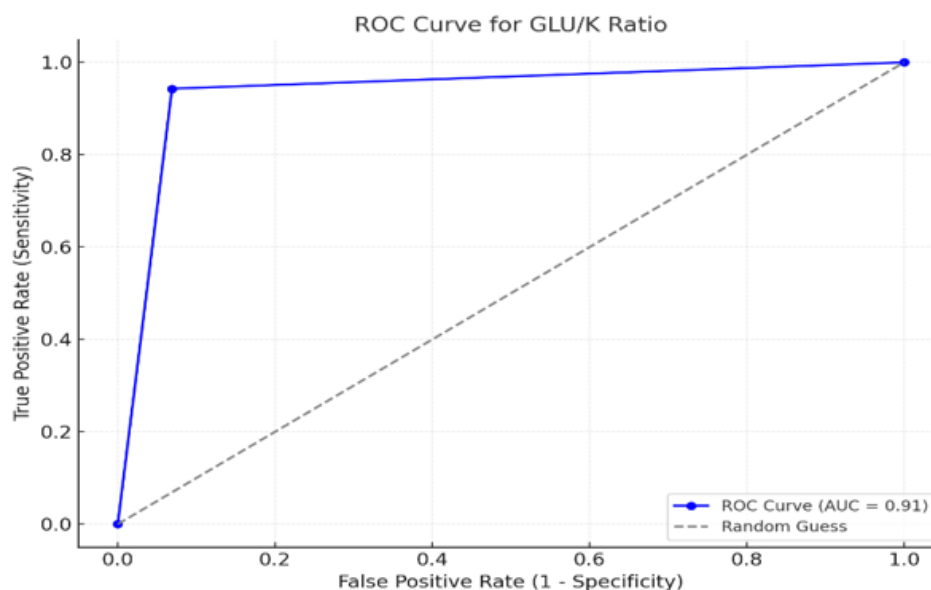


Figure 3: Receiver Operating Characteristic (ROC) curves of GLU/K ratio, K level as an Intermediate Syndrome (IMS) predictor.

The ROC curve for the GLU/K Ratio demonstrates its strong diagnostic performance in predicting Intermediate Syndrome (IMS), with an AUC of 0.910, indicating excellent accuracy. High sensitivity (94.3%) and specificity (93.1%) further support its predictive power. The curve's clear separation from the diagonal highlights its superiority over random classification, reinforcing the GLU/K Ratio's value as a reliable clinical biomarker for IMS.

Table 5: Serum potassium levels in patients with severe organophosphate poisoning

Patient's Serial No.	[K+] (mmol/L)	Muscle weakness or fasciculation	Respiratory distress	Convulsion	Mortality
5	3.9	+	+	-	+
13	2.6	-	-	+	+
27	2.9	+	+	-	-
29	3.9	+	+	-	+
35	3.0	+	+	-	+
39	3.8	+	+	-	-
41	2.8	+	+	-	+
59	3.0	+	-	-	-

61	2.8	+	+	-	+
64	2.5	+	+	-	-
68	3.3	+	+	-	+
72	2.9	+	-	-	-
76	4.0	+	+	+	-
79	2.6	-	+	-	-
91	2.8	+	-	-	+
92	3.9	+	+	-	-
94	2.8	+	+	-	+
99	4.1	-	-	+	+
103	2.8	+	+	-	-
110	2.5	-	+	+	+
112	3.9	+	-	-	+
Total	3.18±0.57	17 (80.95%)	13 (71.43%)	4 (23.81%)	4 (57.14%)

Patient potassium levels ($[K^+]$) are closely linked to clinical symptoms, with muscle weakness and respiratory distress being most common. Convulsions are less frequent (23.81%), indicating limited neurological involvement, while mortality remains high at 57.14%. Hypokalemia (<3.5 mmol/L) is associated with more severe outcomes, including respiratory distress and death. These findings highlight the need for vigilant potassium monitoring and management in acute toxicity cases.

DISCUSSION

Organophosphates (OPs), widely used insecticides in agriculture and households, pose significant health risks, especially in developing countries, due to their role in numerous poisoning cases. OPs inhibit acetylcholinesterase and butyrylcholinesterase, causing acetylcholine accumulation and symptoms like muscle twitching, salivation, and respiratory failure. A severe complication, Intermediate Syndrome (IS), develops 1–3 days post-exposure in 10–40% of survivors, marked by progressive muscle weakness and potential respiratory failure. The serum glucose to potassium ratio (SGPR) is emerging as a predictive biomarker for IS, with higher values indicating greater toxicity. Understanding such markers can aid early risk assessment and improve patient outcomes in OP poisoning (12).

In our study of 228 organophosphate poisoning cases, 9.2% developed Intermediate Syndrome (IMS). Females comprised 43.2% (4.4% IMS) and males 56.8% (4.8% IMS). Most patients were from rural areas (70.8%), with 33.7% being manual laborers and 14.4% farmers. All IMS patients exhibited hypokalemia (mean potassium: 2.8 mmol/L) and died

within 48 hours, precluding confirmation of the typical 3–4 day IMS progression. Monocrotophos was the most common OPC linked to IMS (28%), followed by profenofos (22.7%), malathion (21.7%), chlorpyrifos (19.3%), and dimethoate (8.2%). Despite its lower incidence, dimethoate was linked to the highest mortality due to distributive shock. These findings highlight the fatal synergy between hypokalemia and poisoning severity, supporting Sharif AF et al. (2022), and echo Elmansy AM et al. (2024) and El-Sarnagawy GN et al. (2025) in stressing the toxicological variability among OPCs and the importance of region-specific preventive strategies (13-15).

In our study, dimethoate was the most frequently associated organophosphate in IMS cases (33.3%), followed by malathion (28.5%), monocrotophos and chlorpyrifos (each 14.2%), and profenofos (9.5%). These findings are consistent with Yalçın G et al. (2023), who also identified dimethoate and malathion as major contributors to IMS, likely due to their strong cholinergic toxicity, as noted by Liu J et al. (2024). However, early mortality in IMS patients prevents confirmation of syndrome progression within the typical 3–4-day window (16, 17).

Our study highlighted key differences in OPC distribution between IMS and non-IMS patients. Monocrotophos was most strongly associated with IMS (28%, $p = 0.0048$), while dimethoate dominated in non-IMS cases (33.3%) but was less common in IMS (8.2%). Malathion showed no significant difference (21.7% IMS vs. 28.5% non-IMS). These patterns mirror findings by Boyuk F (2022) and Elmansy AM et al. (2024), linking monocrotophos to higher IMS risk. Regarding exposure types, suicide

was the leading cause of poisoning across both groups (46.8% non-IMS, 52.3% IMS), followed by accidental and occupational exposures. No significant association was found between exposure type and IMS development ($p = 0.277$), consistent with Chen Y et al. (2023) and El-Taftazani EA et al. (2024), who emphasized poisoning severity over cause as a key risk factor. However, early mortality in IMS cases constrained evaluation of typical syndrome progression (14, 18-20).

In our study, vomiting and cough were significantly more common in IMS patients, supporting previous findings by Marini JJ et al. (2023) and Lashin H et al. (2024). Other symptoms showed no significant differences. Most patients with muscle weakness or respiratory distress had hypokalemia (mean ~ 3.0 mmol/L), which was also seen in 23.81% of fatal cases. These results align with Kuo PJ et al. (2024) and Lashin HI et al. (2024), highlighting hypokalemia's link to severe outcomes. Early IMS deaths limited full syndrome assessment (21-23).

Our study demonstrated significantly elevated random blood glucose levels (226.4 ± 80.2 mg/dl) and reduced potassium levels (2.8 ± 0.6 mmol/L) in IMS patients compared to non-IMS patients (120 ± 58.1 mg/dl and 3.5 ± 0.7 mmol/L, respectively; $p < 0.0001$). The GLU/K ratio was markedly higher in the IMS group (82.5 ± 29.8 vs. 40.5 ± 23.8 ; $p < 0.0001$). ROC analysis identified a GLU/K cutoff of 53.2, with 94.3% sensitivity, 93.1% specificity, 62.51% PPV, 93.2% NPV, and an AUC of 0.910 ($p < 0.001$). These results, aligning with Huang CY et al. (2024), Ramadori GP et al. (2023), Klainbart S et al. (2022), and Mohammed E et al. (2023), underscore the GLU/K ratio's strong diagnostic value. However, early mortality among IMS patients restricted evaluation of its predictive power over the typical 3–4-day course (24-27).

CONCLUSION

Our study underscores the significant role of hypokalemia in predicting mortality among organophosphate poisoning patients, with all IMS-positive individuals succumbing within 48 hours, limiting evaluation of typical syndrome progression. Dimethoate was notably associated with the highest fatality, often presenting with distributive shock. Importantly, the glucose-to-potassium (GLU/K) ratio emerged as a reliable and accessible biomarker for early identification of high-risk cases, demonstrating excellent diagnostic performance (AUC: 0.910, $p < 0.001$). These findings emphasize the urgency of early potassium correction and proactive monitoring using GLU/K ratio to improve prognosis and support clinical decision-making in acute OP poisoning management.

REFERENCES

1. Klainbart S, Kelmer E, Sharabany E, Green I, Aroch IJJoVM. Clinical Presentation, Consecutive

- Measurements of Serum Butyryl-Cholinesterase Activity and Treatment of a Dog Intoxicated by Anticholinesterase and Presented Acute Cholinergic Crisis Followed by Intermediate Syndrome. Case Report and Review of the Literature. 2022;77:3.
2. Huang Y-T, Lai P-C, Su C-Y, Chen Y-T, Cai C-Z, Wang C-HJTCMJ. Intermediate syndrome after organophosphate ingestion. 2007;19(3):159-63.
3. Karalliedde L, Baker D, Marrs TCJTr. Organophosphate-induced intermediate syndrome: aetiology and relationships with myopathy. 2006;25:1-14.
4. Saini P, Ochieng IJEAMJ. Intermediate syndrome in organophosphate poisoning: case series. 2016;93(9):466-70.
5. Yang C-C, Deng J-FJotCMA. Intermediate syndrome following organophosphate insecticide poisoning. 2007;70(11):467-72.
6. Baber R, Panay N, Fenton AJC. 2016 IMS Recommendations on women's midlife health and menopause hormone therapy. 2016;19(2):109-50.
7. Alahakoon C, Dassanayake TL, Gawarammana IB, Sedgwick EM, Weerasinghe VS, Abdalla A, et al. Prediction of organophosphorus insecticide-induced intermediate syndrome with stimulated concentric needle single fibre electromyography. 2018;13(9):e0203596.
8. Abdollahi M, Karami-Mohajeri SJT, pharmacology a. A comprehensive review on experimental and clinical findings in intermediate syndrome caused by organophosphate poisoning. 2012;258(3):309-14.
9. Nirmalan N, Nirmalan MJA, Medicine IC. Hormonal control of metabolism: regulation of plasma glucose. 2020;21(11):578-83.
10. Nair PA, Sujatha C. Organic pollutants as endocrine disruptors: organometallics, PAHs, Organochlorine, organophosphate and carbamate insecticides, phthalates, dioxins, phytoestrogens, alkyl phenols and bisphenol A. Environmental chemistry for a sustainable world: Volume 1: Nanotechnology and health risk: Springer; 2011. p. 259-309.
11. Haider R, Mehdi A, Zehra A, Das GK, Ahmed Z, Zameer SJDDR. Drug Development Research in Women. 2024;2(5).
12. Jaga K, Dharmani CJRpdp. Sources of exposure to and public health implications of organophosphate pesticides. 2003;14:171-85.
13. Sharif AF, Fayed MMJN. Assessment of the serum glucose/potassium GLU/K ratio as a predictor of intermediate syndrome following acute anticholinesterase exposure. 2022;89:161-73.
14. Elmansy AM, Hannora DM, Khalifa HKJTR. Serum glucose/potassium ratio as an indicator of early and delayed outcomes of acute carbon monoxide poisoning. 2024;13(5):tfae168.
15. El-Sarnagawy GN, Hafez AS, Ghonem MMJTR. Role of serum glucose/potassium ratio in assessing poisoning severity and adverse outcomes in patients with acute aluminum phosphide poisoning. 2025;101947.
16. Yalçın G, Tunca H, Sayınbatur B, Anıl MJTaop. Predictive value of complete blood count, venous blood gas measurements, and glucose/potassium ratio for delayed neuropsychiatric syndrome in children with acute carbon monoxide poisoning due to coal-burning stove. 2023;58(3):328.

17. Liu J, Luo F, Guo Y, Li Y, Jiang C, Pi Z, et al. Association between serum glucose potassium ratio and mortality in critically ill patients with intracerebral hemorrhage. 2024;14(1):27391.
18. Boyuk FJC, Thrombosis/Hemostasis A. The predictor potential role of the glucose to potassium ratio in the diagnostic differentiation of massive and non-massive pulmonary embolism. 2022;28:10760296221076146.
19. Chen Y, Peng Y, Zhang X, Liao X, Lin J, Chen L, et al. The blood glucose-potassium ratio at admission predicts in-hospital mortality in patients with acute type A aortic dissection. 2023;13(1):15707.
20. El-Taftazani EAE, Eweda SAJZJoFM, Toxicology. Serum glucose potassium ratio compared to theophylline level as a predictor of severity and outcome in acute theophylline toxicity. 2024;22(2):160-78.
21. Marini JI, Sein MEJKJoN. The role of the glucose potassium ratio in the management of traumatic brain injury. 2023;19(1):82.
22. Lashin HI, Sobeeh FG, Sobh ZKJH, Toxicology E. Development and validation of a nomogram for predicting mechanical ventilation need among acutely intoxicated patients with impaired consciousness. 2024;43:09603271241267214.
23. Kuo P-J, Huang C-Y, Hsu S-Y, Hsieh C-HJH. Evaluating the prognostic value of the stress index in trauma patients. 2024;10(17).
24. Huang C-Y, Chou S-E, Huang C-Y, Tsai C-H, Hsu S-Y, Hsieh C-HJD. Role of the stress index in predicting mortality among patients with traumatic femoral fractures. 2024;14(14):1508.
25. Ramadori GPJJoMS. Organophosphorus poisoning: Acute respiratory distress syndrome (ARDS) and cardiac failure as cause of death in hospitalized patients. 2023;24(7):6658.
26. Klainbart S, Kelmer E, Chai O, Segev G, Aroch IJTVJ. Prevalence, clinical manifestations, laboratory findings, treatment, and outcome of intermediate syndrome in anticholinesterase pesticide intoxication of dogs: A retrospective study. 2022;287:105883.
27. Mohammed E, Ali A, Abd El Wahab M, Mahmoud MJASJoFM, Toxicology C. The Potential Role Of Glial Fibrillary Acidic Protein In Evaluation of Organophosphorus-Induced Neurotoxicity: A prospective clinical study. 2023;40(1):101-11.