“EFFECTIVENESS OF SERUM GLUCOSE/POTASSIUM RATIO AS A TOOL FOR PREDICTOR OF INTERMEDIATE SYNDROME”

BY

DR.VISHNU J NAIR

DISSERTATION SUBMITTED

TO

BLDE (DEEMED TO BE UNIVERSITY)
SHRI B. M. PATILMEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPUR – 586103

IN PARTIAL FULFILLMENT FOR THE DEGREE OF

DOCTOR OF MEDICINE IN EMERGENCY MEDICINE

Under the Guidance of

DR. BABU P KATTIMANI

M. S GENERAL SURGERY

PROFESSOR

DEPARTMENT OF EMERGENCY MEDICINE

SHRI B.M PATIL MEDICAL COLLEGE & HOSPITAL &

RESEARCH CENTRE

B.L.D.E. (DEEMED TO BE UNIVERSITY)

SHRI B.M PATIL MEDICAL COLLEGE & HOSPITAL & RESEARCH CENTRE

B.L.D.E. (DEEMED TO BE UNIVERSITY)

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation/thesis entitled "**Effectiveness of serum glucose/potassium ratio as a tool for predictor of intermediate syndrome"** is a bonafide and genuine research work carried out by me under the guidance of **DR. BABU P KATTIMANI** Professor, Dept of Emergency Medicine, Shri B.M. Patil Medical College, Vijayapura, Karnataka.

Date:

Place: Vijayapura

DR.VISHNU J NAIR

Post Graduate student**,**

Department of Emergency Medicine.

BLDE (Deemed to be University)

Shri B M Patil Medical College, Hospital

And Research Centre, Vijayapura

SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL& RESEARCH CENTRE VIJAYAPUR

B.L.D.E. (DEEMED TO BE UNIVERSITY)

CERTIFICATE BY THE GUIDE

This is to certify that the dissertation entitled "**Effectiveness of serum glucose/potassium ratio as a tool for predictor of intermediate syndrome"** is a bonafide genuine research work carried out by **DR.VISHNU J NAIR**. In partial fulfillment of the requirement for the degree of MD in Emergency Medicine.

Date:

Place:- Vijayapura

DR. BABU P KATTIMANI
GUIDE
PROFESSOR
Department of Emergency Medicine
Shri B.M Patil Medical College & Hospital
BLDE Deemed To Be University,
Vijayapura, Karnataka

SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPUR

B.L.D.E. (DEEMED TO BE UNIVERSITY)

ENDORSEMENT BY THE HOD & PRINCIPAL / HEAD OF THE INSTITUTION

This is to certify that the dissertation entitled "**Effectiveness of serum glucose/potassium ratio as a tool for predictor of intermediate syndrome."** is a bonafide research work done by **DR.VISHNU J NAIR** under the guidance of **DR. BABU P KATTIMANI**Professor, Department of Emergency Medicine, Shri B.M. Patil Medical College, Vijayapura.

Seal & Signature of

In Charge HOD of Emergency Medicine

DR Udaykymar J Khasage

M.D (Emergency Medicine)

BLDEU's Shri B.M. Patil

Medical College, Hospital & Research Centre, Vijayapura

Seal & Signature of

The principal

DR Aravind V Patil

M.S (General Surgery)

BLDEU's Shri B.M. Patil

Medical College, Hospital & Research Centre, Vijayapura

Date:

Place: Vijayapura

Date:

Place: Vijayapura

SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPUR

B.L.D.E. (DEEMED TO BE UNIVERSITY)

COPY RIGHT DECLARATION BY THE CANDIDATE

I hereby declare that the BLDE University, Karnataka shall have the right to preserve, use and disseminate this dissertation/thesis in print or electronic format for academic / research purposes.

Dr. Vishnu J Nair
Post Graduate Student,
Department of Emergency Medicine,
Shri B.M. Patil Medical College, Vijayapura.

Date:
Place: Vijayapura

ACKNOWLEDGEMENT

I have got wordless to express my deep sense of gratitude and regards to my guide **DR. UDAYKYMAR J KHASAGE,** Associate Professor and In Charge HOD of Emergency Medicine, under whose inspiring guidance & supervision, I am studying and continuing to learn the art of medicine. His deep knowledge, devotion to work, and zeal for scientific research make him a source of inspiration not only for me but for others too. It is because of his generous help, expert and vigilant supervision, that has guided & helped me to bring out this work in the present form.

My sincere thanks are due to **DR. ARVIND PATIL,** Principal, Shri B.M. Patil Medical College, Vijayapura for permitting me to conduct this study.

I wish to acknowledge and express my gratitude to **DR. BABU P KATTIMANI,** MS Professor, Department of Emergency Medicine sincere thanks to sir for his expert and vigilant supervision and timely advice who have enriched me with their knowledge and experience.

My sincere thanks to all the staff members of Department of Emergency Medicine, Shri B M Patil Medical College Hospital and Research Centre, Vijayapura. I am thankful for the support extend by my senior and junior colleagues.

I would be failing in my duty, if I would not acknowledge my thanks to all the patients and their attenders who were kind enough to help for this study.

I would also like to thank my parents without their constant encouragement & moral support; my studies would have been a distant dream.

I would like to express appreciation to my seniors and juniors who spent time and were always present for my support

DR. VISHNU J NAIR

ABSTRACT

Introduction: Intermediate syndrome (IMS) is a delayed neuromuscular complication of organophosphate (OP) poisoning, developing 24–96 hours after acute cholinergic crisis. It manifests as muscle weakness, particularly affecting the proximal muscles, neck flexors, and respiratory muscles, leading to potential respiratory failure. The precise pathophysiology remains unclear, but persistent acetylcholinesterase inhibition and neuromuscular dysfunction are key contributors.

Objective: The study will evaluate the value of the predictive potential of serum glucose/potassium ratio among patients with at-risk potential of IMS development after OP poisoning.

Materials and Methods: Prospective observational study was performed at Shri B M Patil Medical College with an enrollment of 228 patients of acute OP poisoning. The patients were clinically and biochemically evaluated, and serum glucose and potassium were measured at admission. G/K ratio was performed, and how its creation takes place regarding IMS was related by statistical correlation using ROC curve analysis.

Results: Out of 228 patients, IMS was found in 21 (9.2%). G/K ratio was significantly higher in IMS-positive patients (82.5 ± 29.8) than in IMS-negative patients (24.7 ± 12.6) ($p<0.0001$). ROC analysis provided AUC as 0.910 with cutoff as 53.2 with high sensitivity (94.3%) and specificity (93.1%) for the prediction of IMS.

Conclusion: Serum G/K ratio is a good and valid predictor of IMS in OP poisoning that facilitates early identification of high-risk patients. Its implementation into clinical routines can enhance monitoring and intervention methods and minimize the risk of critical complications.

Keywords: Organophosphate Poisoning, Intermediate Syndrome, Respiratory Failure, Acetylcholinesterase Inhibition, Neuromuscular Dysfunction, Predictive Biomarker, Ratio of Serum Glucose/Potassium.

LIST OF ABBREVIATIONS

- **AUC** - Area Under Curve
- **CKD** - Chronic Kidney Disease
- **CS** - Chi-Square
- **DBP** - Diastolic Blood Pressure
- **d** - Margin of Error
- **ED** - Emergency Department
- **GCS** - Glasgow Coma Scale
- **GLU/K** - Glucose/Potassium Ratio
- **Hb** - Hemoglobin
- **HR** - Heart Rate
- **IMS** - Intermediate Syndrome
- **IV** - Intravenous
- **K+** - Potassium Ion
- **LFT** - Liver Function Test
- **mg/dL** - Milligrams per Deciliter
- **min-max** - Minimum-Maximum
- **mmHg** - Millimeters of Mercury
- **mmol/L** - Millimoles per Liter
- **Na+** - Sodium Ion
- **NPV** - Negative Predictive Value
- **NS** - Not Significant
- **n** - Sample Size
- **OP** - Organophosphate
- **OPC** - Organophosphorus Compound

- **OR** - Odds Ratio
- **p** - Population Proportion
- **p-value** - Probability Value
- **PPV** - Positive Predictive Value
- **q24h** - Every 24 Hours
- **q6h** - Every 6 Hours
- **RBC** - Red Blood Cell
- **ROC** - Receiver Operating Characteristic
- **RR** - Respiratory Rate
- **SBP** - Systolic Blood Pressure
- **SD** - Standard Deviation
- **SPSS** - Statistical Package for the Social Sciences
- **t-test** - Student's t-test
- **USG** - Ultrasonography
- **WBC** - White Blood Cell
- **Z-score** - Standard Score

TABLE OF CONTENTS

Sl No.	Contents	Page No.
1	**INTRODUCTION**	**1-10**
2	**AIMS AND OBJECTIVES**	**11**
3	**REVIEW OF LITERATURE**	**12-47**
4	**METHODOLOGY**	**48-51**
5	**RESULTS**	**52-63**
6	**DISCUSSION**	**64-69**
7	**SUMMARY AND CONCLUSION**	**70-72**
8.	**REFERENCES**	**73-90**
9	**ANNEXURES**	**91-104**
	1. ETHICAL CLEARENCE CERTIFICATE	
	2. CONSENT FORM	
	3.PROFORMA	
	4.MASTER CHART	

LIST OF TABLES

SI No	PARTICULARS	Page No
1.	Demographic features	52
2.	By substance type, the percentage and distribution of individuals with acute exposure to Organophosphorus Compounds (OPCs) (Intermediate Syndrome IMS (-)	53
3.	By substance type, the percentage and distribution of individuals with acute exposure to Organophosphorus Compounds (OPCs) (Intermediate Syndrome IMS (+)	54
4.	Comparison between patients diagnosed with intermediate syndrome IMS and those without intermediate syndrome IMS	55
5.	Distribution of causes between IMS and without IMS group	56
6.	Comparison between patients diagnosed with intermediate syndrome IMS and those without intermediate syndrome IMS related to the main presenting complaints	58
7.	Serum potassium levels in patients with severe organophosphate poisoning	60
8.	Comparison between patients diagnosed with intermediate syndrome IMS and those without intermediate syndrome IMS related to the main presenting Laboratory complaints	61
9.	Receiver Operating Characteristic (ROC) curves, sensitive's and specificities of the significant Intermediate Syndrome IMS predictors.	62

LIST OF FIGURES

Sl No	PARTICULARS	Page No
1	Distribution of patients diagnosed with acute Organophosphorus Compounds OPCs exposure (Intermediate Syndrome IMS (-)) according tothe type of the involved substance	54
2	Distribution of patients diagnosed with acute Organophosphorus Compounds OPCs exposure (Intermediate Syndrome IMS (+)) according to the type of the involved substance.	55
3	Distribution of causes between IMS and without IMS group	57
4	Receiver Operating Characteristic (ROC) curves of GLU/K ratio, K level as an Intermediate Syndrome (IMS) predictor.	63

INTRODUCTION

Intermediate syndrome (IMS) is a delayed neuromuscular sequel after acute organophosphate (OP) poisoning[1]. Organophosphates are ubiquitous in pesticides and insecticides and are capable of inhibiting acetylcholinesterase and producing neuromuscular accumulation of acetylcholine. It leads to hyperstimulation of the cholinergic receptors and presents in three phases: acute cholinergic crisis, intermediate syndrome, and delayed neuropathy[2]. IMS usually occurs 24 to 96 hours after resolution of the acute cholinergic crisis and is associated with weakness in muscles, especially the proximal muscles, neck flexors, and respiratory muscles, and places the patients at extreme risk for respiratory failure. IMS is distinct from the acute cholinergic crisis occurring immediately following exposure and is a condition of hypersecretion of saliva, lacrimation, urination, defecation, and bronchospasm[3]. Whereas the acute phase can be reversed with the administration of atropine and oximes (which reactivates acetylcholinesterase), IMS is a secondary phase of toxicity that is less reversible with these drugs.The precise pathophysiology of IMS remains incompletely understood, but it is believed to be due to dysfunction at the neuromuscular junction, possibly linked to the continued presence of OP compounds or their metabolites[4].

Clinically, patients with IMS exhibit symmetrical muscle weakness, starting with the proximal limbs and the muscles of the neck. Bulbar palsy may develop, leading to dysphagia and difficulty in speech. The most severe complication is respiratory muscle involvement, which can cause hypoventilation and necessitate mechanical ventilation. Cranial nerve function is typically spared in IMS, differentiating it from other neurotoxic conditions [5]. IMS is largely clinical, based on the history of organophosphate exposure, the timing of symptom onset, and the characteristic muscle involvement pattern. Nerve conduction studies may demonstrate decreased compound muscle action potentials that are characteristic of abnormal neuromuscular transmission. IMS is supportive and targets respiratory support in addition to strict monitoring for additional complications. Oximes and atropine function

effectively in acute cholinergic poisoning but are less effective during IMS, and their use remains largely supportive[6]. IMS is resolved in a period of days to weeks and the majority of patients recover complete muscle strength provided they are given proper treatment, though with prolonged respiratory support in the severe ones[7]. Early diagnosis and treatment of IMS are important in order to avert life-threatening respiratory failure. Intermediate syndrome (IMS) following organophosphate (OP) poisoning may be classified into various forms depending upon its clinical manifestation, timing, and severity. Although IMS in most cases is a separate stage of OP poisoning, its heterogeneity has served as the basis for the separation of various types. The types mainly rely on the exposure dose, the chemical organophosphate compound used, the timing of treatment, and patient characteristics like age and underlying medical conditions[8].

Most classification of IMS comes in the form of classifying it as mild, moderate, and severe based on the neuromuscular impairment and impairment of the respiratory apparatus. In mild IMS, there is a weakness of muscles of the shoulder girdle and neck flexors without, or with some minor impairment, of respiratory muscle. They have trouble with maneuvers requiring strength but are not under threat of failure of the respiratory system. With appropriate observation and supportive treatment, mild IMS can be eliminated without the use of invasive therapies[9].

Moderate IMS is associated with more severe muscle weakness, involving trunk muscles and impacting respiration [10]. Initial signs of respiratory distress, including shallow breathing, impaired secretions, and lower oxygen saturation levels, develop in patients. A few require mechanical ventilation, but prognosis is good if early respiratory support is provided. Weakness of bulbar muscles resulting in dysphagia and speech impairment also dominates in moderate IMS.Moderate IMS is characterized by severe weakness of muscles, especially respiratory muscles, which can result in respiratory failure if left untreated[11]. The patients are generally subjected to long-term mechanical support owing to widespread intercostal and diaphragmatic paralysis. Severe IMS may also include the lower cranial

nerves with aspiration and pneumonia complications. Recovering from severe IMS is longer, and even though it is effective in most patients, it is more risky in terms of complications. It is normally noted in organophosphate intoxication with high doses or delayed access to treatment [12].A more nuanced classification designates the early and late-onset IMS, and early-onset IMS is from 24 to 48 hours after exposure, and late-onset IMS from up to 96 hours post-resolution of acute cholinergic toxicity. It is less consistent and potentially also has a prolonged recovery. All of these call for individualized treatment approaches, with the first priority being respiratory support and prevention of further complications, particularly in the moderate and severe presentations[13]. Intermediate syndrome (IMS) of organophosphate (OP) poisoning is caused by a multifactorial interaction of biochemical, toxicological, and patient-related factors. Understanding these factors is necessary for the identification of risk patients and for maximizing management strategies. One of the essential reasons for IMS is chronic inhibition of acetylcholinesterase (AChE), which is an enzyme breaking down acetylcholine at neuromuscular junctions. Organophosphates inhibit AChE and result in a buildup of acetylcholine, which hyper-stimulates cholinergic receptors, particularly at the neuromuscular junction[14]. In the acute process, this presents as cholinergic crisis, but in IMS, the chronic availability of acetylcholine leads to late dysfunctioning of such junctions. The mechanism for this retarded dysfunction is as yet not understood but could involve receptor desensitization or changes in neuromuscular transmission. Organophosphate compound type and dosage are also a factor. The OP compounds have varying potencies and rates of metabolism. AChE binding of some of the compounds with the enzyme forms extremely stable bindings that are less readily reversible even by oxime therapy, aimed at reestablishing AChE function. Second, higher concentrations of OP compounds are associated with increased severity and longer-lasting inhibition of the enzymes as well as increased chance of IMS. The second main parameter is delayed or inappropriate treatment. Intense and timely treatment with antidotes such as atropine and oximes will preclude or lessen IMS severity. Nonetheless, delayed treatment will promote irreversibility of neuromuscular signs which give rise to IMS [15]. In some

instances, despite successful management of the acute cholinergic crisis, the lingering effect of OPs on the neuromuscular system leads to IMS. Factors that include individual patient needs like age, nutritional status, and comorbidities also lead to the production of IMS. Older patients or patients with comorbidities like malnutrition have poor baseline muscle strength and are at higher risk of neuromuscular dysfunction. In addition, liver or renal insufficiency patients also have impaired metabolism and elimination of organophosphate agents, the toxic effect of which is hence extended [16]. Susceptibility of muscle fibers, further, is the second factor responsible. The proximal muscles and respiratory muscles are prone to damage resulting from the long-term overactivation by cholinergic receptors responsible for OP poisoning. The selective susceptibility is the culprit in the characteristic pattern of weakness of the muscles in IMS. IMS is caused by a combination of extended acetylcholinesterase inhibition, delayed treatment initiation, patient vulnerability, and muscle fiber vulnerability, all of which result in its delayed onset and severity[17].

Intermediate syndrome (IM) prediction in organophosphate (OP) poisoning is important for its early treatment and prevention of complications, especially respiratory failure. Various clinical, biochemical, and toxicological predictors of IMS are used to recognize patients at increased risk of developing this delayed neuromuscular syndrome. Acute cholin severity is one of the most important predictors. Patients with marked presentations of the acute cholinergic crisis—i.e., profuse salivation, bronchospasm, seizure, and impairment of consciousness—are at higher risk for IMS development. The severity of the initial response is directly proportional to the extent of inhibition of acetylcholinesterase (AChE) and is a function of the strength of AChE inhibition; extreme AChE inhibition promotes the risk of extensive neuromuscular damage, which subsequently emerges as IMS. The need for mechanical ventilation in the acute stage of poisoning is also a good predictor, signifying severe impairment of respiratory muscles which can either be persistent or recurrent in the intermediate stage. Organophosphate type and dose are significant toxicological predictors. Organophosphates differ with each other in their potency, lipid

solubility, and half-life of action in the body. Those drugs that are lipophilic in nature act with prolonged effects since they get deposited into the fatty cells and then slowly released to the body in small amounts, thereby creating repeated exposure to the toxin and subsequent IMS even though the initial illness has worn off. Higher doses of organophosphates create stronger inhibition of enzymes and higher vulnerability to IMS[18][19][20].

Duration between exposure and treatment is another critical factor. Patients who receive delayed or inadequate initial treatment with antidotes, such as atropine and oximes, are at higher risk of developing IMS. Early administration of these agents can prevent or mitigate the effects of acetylcholine accumulation at the neuromuscular junction. However, if therapy is delayed, the prolonged cholinergic overstimulation can result in neuromuscular junction dysfunction, contributing to the onset of IMS. Studies suggest that patients who do not receive oximes within the first few hours after exposure are more prone to IMS development. Biochemical markers also provide insight into the risk of IMS. Persistent low levels of plasma and red blood cell acetylcholinesterase activity following acute poisoning are associated with an increased risk of IMS. Since acetylcholinesterase inhibition underlies the toxic effects of organophosphates, ongoing suppression of this enzyme indicates that the body has not fully metabolized or cleared the toxin, placing patients at continued risk of delayed complications such as IMS. Patient characteristics such as age, underlying health conditions, and nutritional status can also influence the likelihood of developing IMS. Older patients or those with comorbid conditions, particularly respiratory or neuromuscular disorders, may have a reduced capacity to compensate for the toxic effects of organophosphates. Additionally, malnourished patients may have less muscle reserve and are more vulnerable to the neuromuscular weakness characteristic of IMS. Lastly, prolonged hospitalization or delayed recovery from the acute phase has been noted as a predictor. Patients who take longer to stabilize after the initial phase of OP poisoning are more likely to

experience IMS, reflecting more profound or persistent organophosphate effects on the neuromuscular systemThese predictors establish the need for intensive monitoring and early treatment in organophosphate-exposed patients, especially with initial severe toxicity, delayed therapy, or biochemical evidence of prolonged acetylcholinesterase inhibition[21][22][23].

The serum glucose/potassium ratio has also been proposed as a possible biomarker for the development of intermediate syndrome (IMS) in patients with organophosphate (OP) poisoning. IMS is an early neuromuscular OP poisoning complication that occurs usually 24 to 96 hours after poisoning and may result in death due to respiratory failure if not immediately diagnosed and treated. While clinical severity during the acute cholinergic syndrome and acetylcholinesterase (AChE) inhibition level have been traditional predictors of IMS, recent literature suggests that serum glucose/potassium ratio may provide additional useful prognostic information regarding the likelihood of developing IMS. Organophosphate poisoning results in disruption of the normal metabolic and neuromuscular functions because of the accumulation of acetylcholine as a result of inhibition of acetylcholinesterase. This dysregulation involves more than one organ system, like the endocrine and metabolic functions. Hyperglycemia, i.e., blood glucose elevation, is a typical consequence of OP poisoning as a result of hyperstimulation of the sympathetic nervous system, causing release of catecholamines and glucocorticoids. These hormones are responsible for the stimulation of gluconeogenesis and glycogenolysis with secondary elevation of serum glucose levels. Simultaneously, intracellular potassium shift-induced hypokalemia is a common associate due to disturbed acetylcholine activity leading to hyperstimulation and subsequent insulin release with the forcing of the potassium into the cells. Glucose/potassium ratio encompasses these two metabolic effects in one parameter as a possible expression of systemic overactivity of the cholinergic system. Research on the use of the serum G/K ratio as a prognostic marker for IMS has indicated that the occurrence of an unusually high ratio in the acute phase of OP poisoning can predispose towards a higher

risk of IMS development. Such a correlation may possibly be explained on the strength of the extent of acetylcholine overloading and the attendant metabolic disturbances. For example, an elevated G/K ratio is suggestive of extreme catecholamine hyperglycemia and extreme potassium depletion, both signs that the body is being put under extreme toxic stress[24][25]. What makes the use of the G/K ratio is that it can potentially act as an early and easily quantifiable marker for physicians. Blood glucose and potassium are measured routinely in patients with OP poisoning as a standard part of the workup, and the calculation of the ratio alone increases the workup complexity. High-risk patients can be identified early, by the G/K ratio, which permits intensified monitoring and earlier intervention with possible prevention of the development of severe neuromuscular weakness and respiratory failure. Secondarily, it could also be employed to sort out those patients who would require longer mechanical ventilation or more intensive treatment protocols. Predictive specificity of the G/K ratio, however, has a catch. The ratio may be affected by several conditions not related to OP poisoning like pre-existing diabetes, adrenal insufficiency, renal impairment, or other metabolic derangements. Second, the ratio itself will vary from day to day, depending on when the blood samples are taken, particularly if patients are on intravenous infusions or intravenous insulin therapy. Such variability needs to be taken into account when interpreting tests and is the reason why the G/K ratio cannot be used in isolation, but with other clinical and biochemical markers of risk for IMS, including severity of acute cholinergic poisoning and acetylcholinesterase activity levels. In summary, the glucose-to-potassium ratio is a promising useful, non-invasive technique for the prognosis of the development of intermediate syndrome in organophosphate poisoning[26][27][28].

By adding this metabolic marker to standard diagnostic workups, physicians can enhance their capacity for identifying at-risk patients and managing them in an expedient and targeted fashion to decrease morbidity and mortality of IMS. Nonetheless, validation within larger and more representative populations will be necessary to further establish its predictive value and clinical utility. The G/K ratio encompasses the two metabolic effects and

therefore represents an adequate parameter of the systemic effect of OP poisoning. Elevated ratio, with simultaneous hyperglycemia and hypokalemia, mirrors the degree of the metabolic derangement and cholinergic overactivity that are intimately associated with IMS development. Studies have demonstrated that OP poisonedrole="user","content":"poisoning patients with a high G/K ratio during the acute phase are at higher risk of developing IMS and that this indicates the G/K ratio can be an effective early warning indicator. The effectiveness of the G/K ratio is in being simple and easy to perform.". Routine blood glucose and potassium measurement is also carried out in clinical management of OP poisoning, and hence it doesn't add much complexity to perform the ratio. This parameter can be utilized by doctors to risk-stratify the patients according to IMS and start aggressive monitoring or early intervention like respiratory support in patients with elevated G/K ratio. The predictive function may be helpful in preventing obvious complications such as respiratory failure, a sign of serious IMS. Nevertheless, the effectiveness of G/K ratio as a predictor is based on various parameters such as underlying conditions such as diabetes or renal failure, which may affect glucose or potassium individually[29]. Furthermore, variations in the ratio with measurement time or therapeutic interventions (e.g., fluid or insulin administration) may affect its reliability, whereas the serum G/K ratio is a non-traumatic, valid predictor of IMS risk, it must be employed with other clinical indicators for maximum predictive value. Further studies are needed to fully validate its utility in various patient populations.The serum glucose/potassium (G/K) ratio is another possible good predictor of the development of intermediate syndrome (IMS) in organophosphate (OP) poisoning. As a predictive tool, it has important implications for improved patient outcome and ideal treatment. One of the greatest advantages of the G/K ratio is that it may offer early warning of IMS in patients with OP poisoning. By recognizing individuals at risk prior to full clinical presentation, clinicians are able to intervene earlier to avoid serious neuromuscular complications, including respiratory muscle weakness, which tends to result in mechanical ventilation [30][31][32].

The G/K ratio allows better clinical monitoring as patients with an elevated ratio are at increased risk of developing IMS. More vigilant monitoring of these patients, such as day-and-night respiratory function, muscle power, and neurological status monitoring, enables early identification of deteriorating clinical conditions and reduces complications. The ratio also provides good prognosis. A supra-normal G/K ratio is inappropriately elevated and is associated with greater severity of poisoning and morbidity, and is a marker of the extent of metabolic and cholinergic impairment after OP poisoning. It assists the clinician in the prediction of overall prognosis, i.e., likely duration of stay in hospital and requirement for intensive supportive care.

The G/K ratio is useful in triage and health resource allocation, particularly in those situations where there is a shortage of health resources. In bulk poisoning or in the rural context, the ratio facilitates triaging more severely treated and resource-consuming patients, i.e., ICU admission, in a way that the most severe patients are prioritized first. High G/K ratio patients are advantageously helped by early management[33]. This involves active administration of antidotes such as atropine and oximes, supported by supportive therapy in the form of intravenous fluids, electrolyte replacement (e.g., for hypokalemia), and oxygen therapy. Due to the heightened risk of respiratory muscle weakness, preparation for possible respiratory failure as soon as possible is of utmost significance. Mechanical ventilation or non-invasive respiratory assistance can be instituted based on the risk stratification offered by the G/K ratio. Electrolyte correction is also a crucial component of management since serum potassium derangements play a role in the neuromuscular compromise of IMS. Aggressive and immediate hypokalemia correction may mitigate the severity of IMS as well as overall outcome improvement. Frequent monitoring and rebalancing of serum potassium is a key component of the management regimen. Patients with a raised G/K ratio might require the services of an intensivists, toxicologists, and neurologists with a multidisciplinary team due to the sophistication of the metabolic and neuromuscular complications arising in OP poisoning. Early identification of IMS risk facilitates combined treatment and individualized strategies[34]. Careful follow-up of serum glucose and potassium levels during treatment is necessary because these parameters direct contemporary treatment modalities, for instance, with respect to adjustment of glucose control and electrolyte replenishment. Although the G/K ratio is an encouraging prognostic indicator, underlying conditions such as metabolic disorder, renal impairment, or diabetes mellitus might alter serum levels of glucose and potassium irrespective of OP poisoning and thus make this parameter less important. Thus, the G/K ratio needs to be applied in conjunction with other clinical and laboratory markers for overall risk estimation. In general, the serum G/K ratio is a useful prognostic aid in the prediction of intermediate syndrome development in OP poisoning, allowing early intervention, increased patient monitoring, and optimum use of resources. Its application greatly enhances outcomes by providing guidance that prevents serious complications and allows high-risk patient treatment at an early stage[35].

AIM AND OBJECTIVE

To utilize serum glucose/potassium ratio as an instrument to make a prediction regarding intermediate syndrome of organophosphate poisoning

REVIEW OF LITERATURE

Serum glucose/potassium ratio has also been proposed as a possible predictor of intermediate syndrome (IS), a life-threatening illness accompanying organophosphate (OP) poisoning[36]. Organophosphates, which are widely used as pesticides, inhibit acetylcholinesterase to cause overexposure of nerve endings to acetylcholine. It causes a cascade of events involving the acute cholinergic crisis and, in some cases, intermediate syndrome. IS typically develops 24 to 96 hours after exposure and is characterized by muscle weakness, particularly affecting the respiratory, neck, and limb muscles, often necessitating ventilatory support[37]. Given the substantial morbidity and mortality associated with IS, early detection is critical for timely intervention. Predicting intermediate syndrome remains a challenge as there is no universally accepted biomarker for its early detection. However, the serum glucose/potassium ratio has gained attention due to its physiological relevance in OP poisoning. Organophosphates also interfere with glucose and potassium homeostasis, inducing hyperglycemia and hypokalemia, two of the most important determinants of the neuromuscular manifestations in IS[38]. Hyperglycemia is believed to be secondary to excessive catecholamine release, which enhances glycogenolysis and gluconeogenesis. Furthermore, acetylcholinesterase inhibition can also hinder insulin release, again raising the blood sugar level[39]. Hypokalemia has, however, occurred as a result of intracellular potassium shifts secondary to catecholamine effects and losses through renal and gastrointestinal channels. Glucose/potassium ratio therefore depicts the cumulative metabolic derangement that follows OP poisoning [40]. Prolonged blood glucose and reduced levels of potassium predict the possible onset of intermediate syndrome and as such, makes the ratio a valuable early marker. Also, this ratio can simply be calculated from routine laboratory studies, and therefore it is a valuable and cheap marker for clinical application. Research has indicated that there is correlation between high serum glucose/potassium ratios and the development of IS[41]. Research has indicated that

patients with elevated ratios have greater risk of developing intermediate syndrome than patients with normal ratios, which indicates its predictability. Therefore, it can serve as a prospective indicator. In the clinical environment, the ratio might allow clinicians to detect high-risk patients sooner, with more monitoring, earlier interventions, and potentially improved clinical outcomes. For instance, high glucose/potassium ratio patients might need early ventilatory support or intensive care unit closer observation. Promising as it is, however, the glucose/potassium ratio has its limitations. Further studies are required to establish its usefulness in heterogenous populations and different severities of OP poisoning[42]. Variability between individuals in responses, time of blood sampling, and the particular organophosphate compound could all impact the usefulness of the ratio as a predictor. In addition, absolute cutpoints must be determined to provide consistency and reliability in prediction of IS in different clinical scenarios. Age, comorbidities, and those secondary to complications can influence the discriminatory capacity of the ratio too, and the same need to be controlled in the studies conducted in the future. Serum glucose/potassium ratio has huge potential as an organophosphate poisoning intermediate syndrome predictor[43]. It detects profound metabolic abnormalities in OP toxicity and is a cost-effective, simple approach of early risk stratification. But there must be more intense studies in order to verify its effectiveness and create standardised guidelines on its use to clinical practice. Following further verification, this ratio can serve as an important tool to help in the early diagnosis and treatment of patients with risk of intermediate syndrome, hence creating improved outcomes among patients[44].

Anticholinesterase pesticides are one of the main causes of mortality and morbidity in Egypt. Sharif A et.al;2022 endeavored to assess the serum glucose/potassium (GLU/K) ratio as a risk predictor for intermediate syndrome (IMS) after acute anticholinesterase poisoning. The cross-sectional prospective study was conducted in Tanta University Poison Control Center, Egypt, from January through August 2021 among 243 patients admitted with acute anticholinesterase poisoning. The patients were classified into IMS (+) and IMS (−)

depending on the occurrence of intermediate syndrome. Among the patients, 44 (18.1%) developed IMS, and they were mostly young adults between the ages of 18 years and less than 25 years. Vomiting and abdominal colic were the most frequent symptoms (94.2% and 63.8%, respectively). Significant predictors of IMS included hospital delay, length of stay, mean blood pressure, oxygen saturation, Glasgow Coma Scale score, blood glucose levels, potassium concentration, and the GLU/K ratio. The GLU/K ratio emerged as the most powerful predictor. At a cutoff value of >41.07%, it demonstrated 93% accuracy, 93.2% sensitivity, and 93% specificity in predicting IMS, with an area under the curve of 0.971 ($p < 0.001$). The findings underscore the neurological impacts of organophosphate poisoning beyond its direct muscle toxicity. Doctors should use the GLU/K ratio as a sensitive and early predictive index for IMS to help in early management and intervention of acute anticholinesterase intoxication patients [45].

Carbon monoxide (CO) poisoning is among the major causes of delayed neuropsychiatric syndrome (DNS), which lacks established early biomarkers for predictive reasons. Demirtaş E et.al 2021 assessed serum glucose/potassium (GLU/K) ratio as an easy and convenient predictor of DNS in the emergency department. 281 patients admitted to the emergency department between the years 2012 and 2018. The patients were classified into two groups either having (DNS +) or lacking (DNS −) occurrence of DNS, and the comparison was done between both groups within the GLU/K ratio. From the outcomes, it was noted that the patients in the DNS (+) group showed more increased glucose levels, blood urea nitrogen, carboxyhemoglobin, and GLU/K ratios as compared to the DNS (−) group.The mean glucose levels were 140 ± 34 in the DNS (+) group versus 110 ± 24 in the DNS (−) group ($p < 0.001$), while the GLU/K ratio was 38.35 ± 10.11 versus 28.65 ± 6.53, respectively ($p < 0.001$). The area under the curve for the GLU/K ratio in DNS prediction was 0.791, and a cut point of 35.9 was 63.6% sensitive and 89.6% specific. DNS is a dangerous complication of CO poisoning, and the GLU/K ratio has significant promise as an easy,

immediate initial marker. It can be used to rule out patients who are not at risk of developing DNS, and reserve more intensive care for those at greater risk[46]. Acute type A aortic dissection (ATAAD) is an emergent cardiovascular condition with extremely high surgical mortality. Chen Y et. al;2023 proposed that the serum glucose-potassium ratio (GPR) was related to cerebrovascular disease clinical outcomes and thus might be related to ATAAD outcomes. To investigate, clinical information on 272 patients who underwent surgical treatment for ATAAD from June 2019 to August 2021 was retrospectively analyzed. The patients were separated into two groups according to the median GPR value (1.74), and the findings indicated significantly higher in-hospital mortality in the high GPR group (>1.74) than in the low GPR group (24.4% vs. 13.9%). The incidence of renal dysfunction was also higher in the low GPR group (26.3% vs. 14.8%). Multivariate logistic regression identified independent risk predictors of in-hospital mortality in ATAAD patients as high GPR (>1.74), lactic acid concentration, smoking, and mechanical ventilation. Albumin concentration, on the other hand, was identified as protective predictors for favorable outcomes. GPR >1.74, in particular, had fourfold increased odds of in-hospital mortality (AOR 4.70, 95% CI 2.13–10.40; $P < 0.001$). These observations indicate that GPR would be a good prognostic marker for the identification of high-risk patients after surgical treatment of ATAAD, permitting targeted post-operative care [47].

Acute pulmonary embolism (PE) is a major cause of death and is of two types: massive (MPE) and non-massive (NMPE). The present study was done to evaluate the discriminative utility of the glucose-to-potassium ratio (GPR) between MPE and NMPE. Retrospective analysis was done in 111 patients, of whom 54 were with MPE and 67 were with NMPE. The GPR was compared with the conventional markers such as D-dimer, pulmonary artery pressure (PAP), and C-reactive protein (CRP). The results indicated that D-dimer, CRP, and PAP were high in the MPE group, which indicated a more aggressive course of the disease. The GPR was also high in MPE (30.7 ± 7.5) versus NMPE (24.9 ± 4.3). Boyuk F. et. al; in 2022, GPR was shown to have good diagnostic value with

sensitivity of 72% and specificity of 70% at a cut-off of 26.5. GPR was superior to D-dimer but inferior to PAP, which was the most efficient in diagnosis. The data indicate that GPR might be used as a novel, low-cost marker for MPE/NMPE discrimination as a valuable complement to clinical evaluation, yet must be used together with other more conventional markers such as PAP in solid diagnosis.[48]
High admission serum glucose-to-potassium ratio (GPR) has been associated with poor outcomes in various acute brain injuries, but its prognostic significance in ischemic stroke (IS) was unknown. Lu Y et.al; 2022 had 784 IS patients from a Norwegian emergency cohort and analyzed the relationship between admission GPR and 30-day all-cause mortality. With multivariable logistic regression, the analysis controlled for factors including age, gender, serum sodium, magnesium status, and comorbidities. The analysis showed that higher GPR was independently linked to higher 30-day mortality risk (OR 2.01, 95% CI 1.12, 3.61), implying that GPR can be an important predictor of short-term outcomes in IS patients. Further analysis categorized GPR into tertiles, showing that patients in the highest tertile had a significantly higher risk of mortality compared to those in the lowest tertile (OR 2.15, 95% CI 1.09, 4.24, P for trend = 0.0188). Additionally, a two-piecewise linear regression model demonstrated a clear linear relationship between GPR and 30-day mortality, reinforcing the predictive value of this marker. Overall, GPR at admission may serve as a simple and effective tool for identifying IS patients at higher risk of early death, potentially aiding in early intervention and management strategies[49].

Acute theophylline toxicity poses a significant health risk, particularly in developing countries, due to its severe effects and lack of an antidote. **El-Taftazani EA et.al; 2024** aimed to assess the glucose/potassium ratio as a potential early predictor of the severity and outcome of acute theophylline toxicity, comparing it with serum theophylline levels, which are traditionally considered the gold standard. Conducted on 57 patients admitted to the Poison Control Center of Ain Shams University between January 2021 and June 2022, the

study collected data on sociodemographics, clinical presentation, and laboratory findings, including theophylline concentrations and glucose/potassium ratios. Patients were categorized into minor, moderate, and severe toxicity groups using the Poisoning Severity Score. Both theophylline levels and glucose/potassium ratios were significantly elevated in patients with moderate and severe toxicity compared to those with minor toxicity. Although theophylline levels were more sensitive to the diagnosis of severe cases, the glucose/potassium ratio was more sensitive. It was concluded that an elevated glucose/potassium ratio can be employed as a good marker for early prediction of severe acute theophylline toxicity, an easy and readily available alternative in emergency situations [50].

Methylxanthines are widely used for the management of pulmonary disorders, especially in developing countries, but their therapeutic index is very high to induce morbidity and mortality. Sharif AFet.al; 2022 tried to assess the glucose/potassium ratio as an early indicator of life-threatening events (LTEs), i.e., cardiovascular and neurological diseases, in acute methylxanthine poisoning patients. Performed as a two-year retrospective cohort study at an Egyptian Poison Control Center, the study examined medical histories of 366 patients, of which 59 (16.1%) had LTEs, the most frequent severe arrhythmia seen being T wave inversion. The study identified a number of laboratory parameters, such as random blood glucose and potassium level, glucose/potassium ratio, and others, to be highly predictive of LTEs. Of these, glucose/potassium ratio had the highest prediction with an odds ratio of 2.92 and 95% confidence interval of 2.02–4.23. The ratio had an excellent area under the curve of 0.906, reflecting excellent diagnostic accuracy, and the cut-point value of 2.44 gave 88% sensitivity and 70% specificity. Generally speaking, the outcome prefers the glucose/potassium ratio as an effective and available predictor for sorting persons according to LTE severity and danger, useful for clinical decision and optimization of treatment for victims of methylxanthine intoxication.

Bouida W et.al; 2017 discusses the effect of intensive insulin therapy with glucose-insulin-potassium (GIK) on hospitalized patients with acute coronary syndrome (ACS) during one year with special focus on whether the hyperglycemia caused by GIK infusions would affect such effects. 772 patients with non-ST-segment elevation ACS were randomly allocated to three groups: the GIKI2 group with GIK and intensive insulin therapy, the GIK group with non-intensive insulin therapy, and a control group with standard care. The main outcome was the rate of major cardiovascular events, death, reinfarction, and stroke, at one year. Results showed that the GIKI2 group demonstrated significantly lower frequency of severe cardiovascular events (12.8% vs. controls, 20.5%) to propose an advantageous impact of intensified insulin therapy with GIK. Modulated platelet activity and fibrinolysis markers consistent with these results were further observed by the study. The GIKI2 group specifically showed lower platelet activity and significant lowering of plasminogen activator inhibitor-1 levels that enhance fibrinolysis. In contrast, the control group developed augmented platelet reactivity and higher plasminogen activator inhibitor-1 levels. Despite having a greater incidence of mild hypoglycemia in the GIKI2 group, the overall findings suggest that aggressive insulin therapy with GIK enhances one-year outcome in non-ST-segment elevation ACS patients, predicting its future ability to enhance clinical outcomes through modification of platelet activity and enhancement of fibrinolysis[51].

Traumatic brain injury (TBI) is a severe public health problem, especially in setting proper initial management protocols at hospitalization, such as the need for surgery. Marini JI et.al; 2023 explains the use of the glucose-to-potassium ratio as a prognostic biomarker of patient outcomes in TBI, considering its simplicity and very high accuracy compared to other biomarkers. The study included patients treated at a single center during 2020-2021, with an emphasis on those diagnosed with mild TBI necessitating neurosurgery, and moderate and severe TBIs. Blood samples were collected at admission, and the glucose-to-potassium ratio was calculated and correlated with patient outcomes at six and twelve months. A total of 47 patients met the study criteria, with 35 (74%) achieving favorable outcomes and 12 (26%)

experiencing poor outcomes. Among the various biomarkers assessed, only the glucose-to-potassium ratio demonstrated a significant association with outcomes in both bivariate and multivariate analyses (p=0.04; odds ratio, 8.61; 95% confidence interval, 1.07–69.6). This indicates that a higher glucose-to-potassium ratio correlates with poorer outcomes and increased mortality among TBI patients. The findings underscore the glucose-to-potassium ratio's potential as a valuable tool in clinical practice for predicting TBI outcomes, highlighting its importance in guiding early management decisions[52].

Delayed neuropsychiatric syndrome can arise after carbon monoxide poisoning, even after apparent recovery, yet research on predictive indicators in pediatric patients is scarce. This study aimed to evaluate the effectiveness of various hematological and biochemical parameters—such as complete blood count metrics, neutrophil/lymphocyte and platelet/lymphocyte ratios, systemic immune inflammation index, glucose/potassium ratio, venous blood gas parameters, and carboxyhemoglobin levels—in predicting this syndrome in children exposed to carbon monoxide from coal-burning stoves. **Yalçın G et.al;2013** involved 137 pediatric patients admitted with acute carbon monoxide poisoning between 2014 and 2019, with 46 diagnosed with delayed neuropsychiatric syndrome within a year. Significant differences were found in several blood parameters among the groups ($P < 0.05$). The most effective predictors identified were the systemic immune inflammation index (area under the curve = 0.852; sensitivity = 89.1%; specificity = 75.8%), neutrophil count (AUC = 0.841; sensitivity = 78.2%; specificity = 79.1%), and the neutrophil/lymphocyte ratio (AUC = 0.828; sensitivity = 78.2%; specificity = 75.5%). Approximately one-third of children who experienced carbon monoxide poisoning developed delayed neuropsychiatric syndrome, highlighting the importance of these early predictors in clinical settings[53].

This prospective cohort study compared outcomes between patients whose serum potassium (K+) levels were 3.5–4.5 mEq/L with those of patients who had abnormal levels.

This study was carried out during May 2012 to February 2013, Tongyoo S et. al; 2018 enrolled 160 patients whose baseline data, Acute Physiology and Chronic Health Evaluation II (APACHE II) scores, serum K+ levels, and hospital outcomes were documented. The patients were assigned to a normal K+ group (mean K+ between 3.5 to 4.5 mEq/L) and an abnormal K+ group (mean K+ values outside the range above). The findings revealed 74 patients (46.3%) to be in the normal K+ category, while the abnormal K+ category showed a much greater APACHE II score, more frequency of coronary artery disease, and more vasopressor use. Additionally, abnormal serum K+ values pointed to a greater risk of mortality in the ICU and higher incidence of ventricular fibrillation as opposed to normal K+ levels. These results indicate that critically ill patients with an abnormal potassium level are at increased risk of developing severe cardiac complications and elevated mortality in the ICU[54].

Wahab NN et.al; 2002 attempted to assess the prognostic value of hyperglycemia in acute myocardial infarction (AMI) patients during the thrombolytic era according to contemporary definitions of hyperglycemia. Previous studies employed different definitions of hyperglycemia and were performed prior to the initiation of disease-modifying therapy. The study comprised 1,664 consecutive AMI patients admitted from a population-based register between October 1997 and October 1998. The patients were divided according to history of diabetes and whether their blood glucose concentration was greater than 198 mg/dl (11 mmol/l). Analysis of the effect of different cardiac risk factors, interventions, and treatments on mortality employed multivariate logistic regression to investigate the association of blood glucose concentrations with patient outcome. The results indicated that among nondiabetic patients, their in-hospital mortality was considerably higher (odds ratio 2.44) among those with blood glucose levels more than 198 mg/dl compared to lower levels. In diabetic patients, the same held true. The findings reinforce that hyperglycemia among patients with AMI, even non-diabetic, is associated with worse outcomes. This underlines the importance of

tight glucose control and points towards enhanced screening for diabetes to enhance patient care in acute care [55].

Zhang L et.al 2005 studies the influence of a metabolic intravenous glucose-insulin-potassium (GIK) cocktail on plasma concentrations of soluble Fas/APO-1 (sFas) and Fas ligand (sFasL) in patients receiving reperfusion therapy for acute myocardial infarction (AMI). Earlier clinical research demonstrated that GIK might decrease mortality in AMI patients, and earlier studies suggested that apoptosis mediated by Fas is responsible for ischemic/reperfusion injury. The research involved 74 AMI patients who were assigned randomly to either a high-dose GIK infusion (n=35) for 24 hours or a placebo infusion (n=39). A control group of 34 patients was involved. The results showed that the levels of sFas and sFasL in AMI patients were significantly higher than normal controls. After GIK infusion, sFas levels decreased at 24 hours but reverted to increase from days 3-7 before declining by day 14. Vehicle administration produced no changes. sFasL levels remained the same in both groups throughout the study. These results indicate a correlation of sFas and sFasL levels with AMI and that high-dose GIK therapy is cardioprotective and thus holds promise as an AMI therapy. The sFas might also serve as a good physiological adaptation marker of ischemic/reperfusion injury[56].

The Glucose Insulin in Stroke Trial (GIST-UK) looked at the influence of glucose potassium insulin (GKI) infusion on blood pressure in hyperglycemic patients with acute stroke. Scott JF et.al; 2001 randomly assigned 145 adult patients with plasma glucose 6.1-17 mmol/l to receive GKI infusion (500 ml 10% glucose, 20 mmol potassium chloride, 16 units insulin) or saline control for 24 hours. Glucose levels were measured at every two-hour interval, blood pressure at four-hour intervals, and plasma glucose at eight-hour intervals. In the GKI group, insulin was titrated to keep the glucose between 4 and 7 mmol/l. Systolic blood pressure was significantly lower in the GKI group between 4 to 24 hours except at 8 hours. Neurological impairment was assessed using the European Stroke Scale (ESS), and no between-group difference in total ESS sum was observed on day 7, but much better

recovery of the GKI group than on admission was observed on day 7. The study concluded that GKI treatment in acute stroke significantly decreased systolic blood pressure without causing neurological deterioration and proposed that it might be effective for stroke management[57].

This study tested for correlation between the serum glucose concentration at admission and outcome in 1,446 consecutive acute ischemic strokes. Admission serum glucose was measured simultaneously with other clinical and radiological parameters. NIHSS score at 24 hours and Rankin score at 3 and 12 months were noted. Serum glucose in univariate as well as multivariate analysis was an independent predictor for 12-month functional outcome (OR 1.15, P=0.01). Other predictors that were significant included admission NIHSS score, age, prestroke Rankin score, and leukoaraiosis. Ntaios G et.al; 2010 reported a J-shaped relationship between stroke outcome and serum glucose, with an ideal nadir at 5 mmol/L. Glucose levels of 3.7 to 7.3 mmol/L were linked with good outcomes, such as Rankin score ≤2 at 12 months. Likewise, glucose levels of 4.0 to 7.2 mmol/L were linked with 24-hour NIHSS score of <7. This research indicates that both hyperglycemia and hypoglycemia are detrimental in acute ischemic stroke, highlighting the importance of keeping glucose within 3.7-7.3 mmol/L for improved outcomes [58]. This study assessed the impact of admission blood glucose level (BGA) and 48-hour glucose change (CG48) on outcome in diabetic and nondiabetic stroke patients with acute ischemic stroke (AIS) treated with endovascular therapy. There were 614 patients in 7 US centers (2006–2009), and these included demographics, risk factors for stroke, and 90-day clinical outcomes (death and modified Rankin Scale score of 3–6). Natarajan SK et.al; 2011 observed that in nondiabetic patients, BGA ≥116 mg/dl (≥6.4 mmol/L) and the inability of glucose to fall by >30 mg/dl (>1.7 mmol/L) at 48 hours were highly predictive of adverse outcomes and mortality ($p < 0.001$). In diabetic patients, only the BGA ≥116 mg/dl was an independent predictor of poor outcomes ($p = 0.001$), while CG48 did not influence outcomes. The researchers developed the BRANCH scale, which combines BGA, Thrombolysis in

Myocardial Infarction (TIMI) Grade 2–3 reperfusion, age, NIHSS score at presentation, CG48, and symptomatic intracranial hemorrhage to predict 90-day outcomes. The scale demonstrated strong predictive accuracy (AUC > 0.79) and holds potential as a simple prognostic tool for AIS patients undergoing endovascular therapy, though it requires prospective validation [59].

Potassium testing is crucial in emergency medicine, and a quick turnaround time (TAT) enhances emergency department efficiency. TAT refers to the time between a laboratory receiving a specimen and releasing the test report. Shortening TAT typically requires significant resources, training, and time, so this study aimed to find a convenient way to reduce TAT and identify factors that affect the timeliness of emergency potassium test reports. **Lv S et.al; 2024** analyzed factors such as sex, age, potassium levels, the number of tests, specimen processing time, instrument status, critical value ratio, and staff work experience. The dependent variable was whether potassium test reports took more or less than 30 minutes to be completed. Multivariate analysis revealed that work experience, instrument failure, and specimen processing time were key factors in delayed reporting. Following the implementation of improvement measures, the time taken for potassium reporting significantly decreased. This study highlights the effectiveness of logistics in reducing TAT for potassium testing in emergency settings, offering a fresh perspective on quality management in the lab. By addressing specific risk factors, emergency departments can achieve more timely potassium reporting, ultimately improving patient care [60].

Chronic heart failure (CHF) is a common comorbidity in critically ill patients in the ICU and is associated with poor outcomes. **Lin Z et.al; 2022** aimed to explore the relationship between the blood urea nitrogen to serum albumin ratio (BAR) and the prognosis of CHF patients in the ICU.

A retrospective cohort of 1,545 patients with CHF from the MIMIC-III database was analyzed. The study found that 27.6% of patients (n = 427) had a 90-day all-cause mortality, and 17.3% (n = 267) had in-hospital mortality. Logistic regression analysis revealed that a higher BAR is an independent risk factor for in-hospital mortality. Compared to patients with BAR ≤ 0.83, those with BAR between 0.83 and 1.24 had an odds ratio (OR) of 2.647 for in-hospital mortality, and those with BAR ≥ 1.24 had an OR of 3.628. Additionally, COX regression analysis showed that higher BAR was associated with increased all-cause mortality at 90 days. Kaplan-Meier curves confirmed these findings, and the area under the ROC curve for predicting in-hospital and 90-day mortality were 0.622 and 0.647, respectively. In conclusion, BAR is a significant independent predictor of in-hospital and 90-day mortality in critically ill CHF patients admitted to the ICU[61].

Acute cholangitis (AC) is a common inflammatory disease and a leading cause of septic shock, often associated with high hospital mortality. Current models for predicting short-term mortality in AC patients are suboptimal. **Pan LN et.al; 2023** aimed to develop a new model to predict 30-day mortality in AC patients. Data from the Medical Information Mart for Intensive Care IV version 2.0 (MIMIC-IV v2.0) included 506 AC cases, split into a 70% training set and a 30% validation set. A multivariate logistic regression was used to create a predictive nomogram for 30-day mortality. The model's effectiveness was assessed using the area under the receiver operating characteristic curve (AUC), calibration curve, net reclassification improvement (NRI), integrated discrimination improvement (IDI), and decision curve analysis (DCA). Of the 506 patients, 14% (71) died within 30 days. Independent risk factors identified included GCS, SPO2, albumin, AST/ALT, glucose, potassium, PTT, and peripheral vascular disease. The nomogram outperformed other common scoring systems, such as SOFA, OASIS, and SAPS II. Calibration curves showed good

alignment between predictions and actual outcomes, and DCA revealed strong clinical utility [62].

The stress index (SI), calculated as the serum glucose-to-potassium ratio, is being explored as a potential prognostic marker in trauma patients. **Kuo PJ et. al; 2024** aimed to assess its predictive value for clinical outcomes, particularly in-hospital mortality, among trauma patients. A retrospective analysis was conducted on 20,040 adult trauma patients admitted to a single trauma center from January 2009 to December 2022. SI was determined using serum glucose and potassium levels recorded upon emergency room arrival. Patients were categorized into two groups based on an optimal SI cutoff, identified via receiver operating characteristic (ROC) curve analysis. Results showed that patients who died had a significantly higher SI (59.7 ± 30.6) compared to survivors (39.5 ± 17.5), with $p < 0.001$. Multivariate analysis identified that SI was an independent predictor of mortality (odds ratio [OR] 4.65, 95% confidence interval [CI]: 2.61–8.27, $p < 0.001$). The group with high SI (≥42.7) also had significantly poor outcomes, with increased in-hospital mortality (7.5% vs. 1.4%, $p < 0.001$) and hospital stays. Therefore, SI is an independent predictor of mortality and poor outcomes in trauma patients and is a good prognostic marker for early risk stratification[63].

Tsivgoulis, G et.al; 2016 assessed the effect of admission hyperglycemia (aHG, ≥144 mg/dL) on outcomes in intravenous thrombolysis (IVT)-treated acute ischemic stroke (AIS) patients, stratified by history of diabetes mellitus (DM). Outcomes were assessed from SITS-ISTR registry data using propensity score matching (PSM). Symptomatic intracranial hemorrhage (SICH) was the main safety outcome, and functional independence (FI) at 3 months (modified Rankin Scale [mRS] 0-2) was the main efficacy outcome. In the non-DM group (12,318 patients), those with aHG had worse 3-month outcomes, with lower FI rates (53.3% vs. 57.9%, $P < 0.001$), higher mortality (19.2% vs. 16.0%, $P < 0.001$), but similar SICH rates (1.7% vs. 1.8%, $P = 0.563$) compared to patients without aHG. Similarly, in the

DM group (6,572 patients), aHG was linked to poorer functional outcomes (mRS 0-1: 34.1% vs. 39.3%, P < 0.001), lower FI (48.2% vs. 52.5%, P < 0.001), higher mortality (23.7% vs. 19.9%, P < 0.001), and no difference in SICH (2.2% vs. 2.7%, P = 0.224). In conclusion, aHG was associated with worse 3-month outcomes in both DM and non-DM AIS patients treated with IVT, affecting mortality and functional recovery without increasing SICH risk[64].

Hyperglycemia (HG) is common in acute stroke and is recognized as a potential therapeutic target due to its association with larger infarct size, poor clinical outcomes, and increased mortality. This has led many stroke centers to implement intensive insulin therapy (IIT) to manage HG, inspired by its use in intensive care units. However, recent evidence has diminished support for IIT in stroke care due to its high risk of inducing hypoglycemia, which may worsen outcomes. The UK Glucose Insulin in Stroke Trial (GIST-UK), involving 933 patients, found no clinical benefit from IIT in stroke management, while smaller trials consistently showed a high risk of hypoglycemia but failed to demonstrate significant clinical advantages. **Piironen K et. al; 2012** , there is a growing consensus that safer methods for managing glucose in stroke patients are needed before conducting large randomized trials. Additionally, understanding HG's complexity in acute stroke is crucial, as it can result from factors like known or undiagnosed diabetes, metabolic syndrome, stress response, or the stroke lesion itself. Further experimental work and human studies are required to explore new strategies for glucose control and to better understand HG's toxicity in the context of acute stroke[65].

A significant number of acutely intoxicated patients present with impaired consciousness, and identifying those who may require advanced care, such as mechanical ventilation (MV), is crucial for improving outcomes. **Lashin HI et.al;2024**analyzed 330

acutely intoxicated patients admitted to Tanta University Poison Control Center in Egypt from January 2021 to December 2023. The patients were divided into a derivation cohort (257 patients) and a validation cohort (73 patients) to develop and validate a predictive nomogram for determining the likelihood of MV necessity. The strongest predictors of need for MV were mean arterial blood pressure (OR = 0.96), PaO2 (OR = 0.96), pH (OR = 0.00), and glucose/potassium ratio (OR = 1.59). The predictors helped to derive a bedside nomogram, which was extremely predictive with an area under the curve (AUC) of 95.7%, accuracy of 93.4%, sensitivity of 88.9%, and specificity of 95.1% in ROC analysis.External validation also demonstrated the efficacy of the nomogram with an AUC of 96.5%. The validated model is beneficial to clinicians practicing in low-resource environments and can predict patients requiring MV early, thereby enhancing patient management and outcomes [66].

The Stress Index (SI), which is defined as the blood glucose to serum potassium ratio, has the potential to be used as a prognostic indicator in acute care, in particular for mortality prediction among isolated moderate-to-severe traumatic brain injury (TBI) patients. This was a retrospective cohort of 4,357 adult patients presenting with isolated moderate to severe TBI who were treated from 2009 through 2022. SI was calculated using initial glucose and potassium levels upon emergency department arrival. Logistic regression models assessed the relationship between SI and mortality, adjusting for relevant variables, and receiver operating characteristic (ROC) analysis determined the optimal SI threshold for predicting mortality. **Huang CY et. al; 2024** studied, 463 (10.6%) died, with deceased patients exhibiting a significantly higher SI (61.7 vs. 44.1, $p < 0.001$). Multivariate analysis revealed that a higher SI independently predicted an increased mortality risk (odds ratio [OR] 6.70, 95% confidence interval [CI] 1.66–26.99, $p = 0.007$). The optimal SI cutoff for mortality prediction was identified as 48.50, demonstrating sensitivity of 62.0% and specificity of 71.4%. Patients with an SI $\geq$ 48.5 had nearly double the adjusted odds of mortality compared to those below

this threshold. Therefore, SI could be integrated into clinical assessments to improve risk stratification and management for TBI patients [67].

Traumatic femoral fractures, often resulting from high-energy incidents like traffic accidents, require prompt management to prevent severe complications. **Huang CY et.al; 2023** investigates the prognostic significance of the Stress Index (SI)—the ratio of glucose to potassium—in patients with these fractures. The retrospective cohort study examined adult trauma patients aged 20 and above with traumatic femoral fractures from a level 1 trauma center in southern Taiwan, covering the period from January 1, 2009, to December 31, 2022. Serum electrolyte levels were assessed upon emergency room arrival, and the SI was calculated. Among the 3,717 patients analyzed, 64 died, with deceased patients showing significantly higher blood glucose (199.3 vs. 159.0 mg/dL, $p < 0.001$) and SI levels (53.1 vs. 41.6, $p < 0.001$). The optimal SI cutoff for predicting mortality was determined to be 49.7, with sensitivity of 53.1% and specificity of 78.7% (AUC = 0.609). Higher SI levels were associated with increased mortality (4.2% vs. 1.0%, $p < 0.001$) and longer hospital stays (12.8 vs. 9.5 days, $p < 0.001$). Adjusted odds ratios indicated that a higher SI significantly increased the risk of mortality (AOR 2.05, $p = 0.016$). While the SI demonstrates moderate predictive value, it serves as a valuable early risk assessment tool, warranting further prospective studies for validation [68].

Neurocognitive disorders (NCDs) and sleep disturbances are common in the perioperative and intensive care unit (ICU) settings, yet there is a lack of tailored assessment tools for critically ill patients with these conditions. **Li Y et. al; 2022** aimed to develop and validate prediction models for NCDs among adult patients experiencing sleep disturbances. Utilizing the MIMIC-IV database, researchers analyzed data from adult ICU patients diagnosed with sleep disturbances based on ICD-9 and ICD-10 codes. They employed logistic regression and LASSO analyses to identify key risk factors for NCDs and create

nomograms for prediction. The models' performance was assessed using bootstrap resampling, receiver operating characteristic (ROC) metrics, area under the ROC curve (AUC), and decision curve analysis (DCA). Ten significant risk factors were identified, including age, gender, midazolam use, morphine use, glucose levels, diabetes, potassium levels, international normalized ratio, partial thromboplastin time, and respiratory rate. The logistic regression model achieved a sensitivity of 74.1% and specificity of 64.6%. The LASSO model with the addition of platelet count and Glasgow Coma Score and the exclusion of cardiovascular diseases had improved sensitivity (86.1%) and specificity (82.8%). The AUC of the logistic and LASSO models were 0.730 and 0.920, respectively, and had high predictive ability [69].

In-hospital cardiac arrest (IHCA) is associated with poor prognosis and low survival discharge. Although several prognostic markers, including interleukin-6 and high sensitivity C-reactive protein, have been evaluated in outcome prediction following return of spontaneous circulation (ROSC), they are not available on a regular basis and could be expensive. Patel VH et. al; 2019 investigates the neutrophil-lymphocyte ratio (NLR) as a potential prognostic indicator for mortality in patients with IHCA who achieve ROSC. Conducted as a retrospective analysis at a large urban community hospital over one year, the study examined patients who underwent advanced cardiac life support following IHCA. Participants were categorized based on their NLR values (NLR < 4.5 or NLR ≥ 4.5), a cutoff determined by receiver operating characteristic curve analysis, which indicated a positive predictive value of 73%, sensitivity of 82%, and specificity of 42% for predicting in-hospital death. Among the 153 patients (mean age 66.1 years, 48% female), 65% experienced in-hospital mortality. Survivors had a median NLR of 4.9, while non-survivors had a median NLR of 8.9 ($P = 0.001$). A multivariable logistic regression revealed that an NLR ≥ 4.5, older age, and elevated serum lactate levels were independent predictors of mortality, suggesting that elevated NLR may effectively indicate increased death risk in IHCA patients [70].

During the first wave of COVID-19, a rapid increase in admissions highlighted the need for an efficient risk stratification tool to assist in patient triage. Currently, no clinical prediction tool specifically exists for COVID-19 hospital admissions. **Fishbein J et.al; 2022**analyzed data from 7098 patients admitted to 13 hospitals within the Northwell Health system in New York from March 1 to April 27, 2020. Inclusion criteria included a positive SARS-CoV-2 nasal swab, a 12-lead ECG within 48 hours, and a complete metabolic panel within 96 hours of presentation. The overall in-hospital mortality rate was 27.1%. Independent predictors of mortality included male gender, older age, low oxygen saturation (<92%), high heart rate (>120 bpm), elevated serum lactate, sodium, blood urea nitrogen, and creatinine levels, as well as comorbidities such as heart failure and chronic obstructive pulmonary disease. Additionally, a corrected QT interval (QTc) longer than 500 ms was identified as an independent risk factor for mortality (OR 1.41). These factors were incorporated into a risk score with an area under the curve (AUC) of 0.78, demonstrating strong predictive accuracy for in-hospital mortality. The study underscores the importance of QTc prolongation as a mortality risk in COVID-19 patients [71].

The belief that stress-induced hyperglycemia in critically ill patients is beneficial has been questioned by recent studies. Two large randomized trials have shown that maintaining normoglycemia through intensive insulin therapy significantly reduces both morbidity and mortality. Despite these findings, concerns remain about the overall efficacy of this approach, especially in certain patient subgroups, as well as the risks associated with hypoglycemia and high-dose insulin use.**Vanhorebeek I et.al; 2007** indicates that intensive insulin therapy can lower hospital mortality by 3-4% in critically ill patients. To confirm this survival benefit with statistical significance, future studies would need larger sample sizes, ideally around 5,000 participants. Moreover, the risk reduction for mortality rises to approximately 8% when insulin therapy is continued for at least three days. Strict blood glucose control appears to be

necessary to achieve the best outcomes. While the risk of hypoglycemia increases with intensive insulin therapy, it is still uncertain whether this poses a significant harm in the context of critical care. Further research is needed to balance the benefits of glucose control with the potential risks of hypoglycemia [72].

Admission hyperglycemia has been linked to worse outcomes in ischemic stroke patients. **Poppe AY et.al; 2009** explored whether hyperglycemia (glucose >8.0 mmol/l) in the early phase of stroke is associated with higher mortality, symptomatic intracerebral hemorrhage (SICH), and poor functional outcomes at 90 days in patients treated with intravenous tissue plasminogen activator (IV-tPA). Data from the Canadian Alteplase for Stroke Effectiveness Study (CASES) involving 1,098 stroke patients were analyzed. Among these, 27% had hyperglycemia on admission, with 18% of non-diabetic and 70% of diabetic patients affected. After adjusting for other factors, hyperglycemia was independently associated with a higher risk of death (1.5 times greater), SICH (1.69 times higher), and lower chances of favorable recovery at 90 days (0.7 times lower). The risk of poor outcomes, including death and SICH, increased as admission glucose levels rose. These associations were consistent in both diabetic and non-diabetic patients. The study concludes that admission hyperglycemia in stroke patients treated with IV-tPA is a significant predictor of worse outcomes, including increased mortality and poorer functional recovery. Further research is needed to determine whether hyperglycemia is a modifiable risk factor for improving stroke outcomes [73].

Refeeding syndrome is a life-threatening condition characterized by electrolyte imbalances, fluid overload, and organ failure, often occurring in individuals with risk factors like starvation, chronic alcoholism, anorexia nervosa, or prolonged fasting after surgery. The syndrome is triggered by excessive or unbalanced nutrition intake, and prevention requires careful identification of at-risk individuals and controlled nutrition support with electrolyte

supplementation. **Rio A et.al; 2013** conducted at a large university hospital from 2007 to 2009, 243 adults receiving artificial nutrition support were monitored. Of these, 133 had risk factors, including a body mass index (BMI) below 18.5, significant weight loss, poor nutritional intake, or a history of substance abuse. Refeeding syndrome developed in 3 patients (2%) who experienced severe electrolyte shifts and organ dysfunction, though no deaths were directly attributed to the syndrome. Overall, 13 participants (5.3%) died during the feeding period, and 68 (28%) died during their hospital stay from unrelated causes like organ failure or respiratory failure. Baseline low-serum magnesium was an independent predictor of refeeding syndrome, and the infusion of carbohydrates before artificial nutrition may have triggered the syndrome in some cases. However, refeeding syndrome was rare and survivable with proper management in at-risk patients[74].

Tan L et. al; 2021 sought to develop and validate a nomogram for the prediction of hospital mortality in acute myocardial infarction (AMI) patients in the ICU within 24 hours of ICU admission. Using data from 2,704 patients in the eICU-Collaborative Research Database (eICU-CRD), univariate and multivariate logistic regression analysis identified 14 predictors. These were some of the predictors like age, peripheral vascular disease, atrial fibrillation, cardiogenic shock, norepinephrine use, urine output, and serum parameters like white blood cells, hemoglobin, glucose, and magnesium. The performance of the nomogram was estimated by different methods like Harrell's concordance index (C-index) of 0.834, receiver operating characteristic (AUC) analysis, calibration curves, and decision curve analysis (DCA), establishing its validity in clinical prediction. Validation in a second cohort of 1,026 patients from the MIMIC-III Database upheld its predictive validity. The AUC of the nomogram (0.885) was superior to conventional ICU scoring models (range, 0.811-0.860). Briefly, the nomogram, derived from 14 variables, is a useful tool for early detection of high-risk AMI patients in the ICU and is superior to conventional ICU scoring models in predicting

hospital mortality. Its good predictive ability and clinical utility make it useful in directing interventions in intensive care[75].

This study will build a predictive model of Enlarged Perivascular Spaces (EPVS) in neurology and neuroradiology, with their relationship mainly to Cerebral Small Vessel Disease (CSVD) and neurodegenerative disease. Li N et. al; 2021 studied 587 neurology inpatients, applying LASSO regression as the feature selection method and logistic regression to build the model. Predictors that were identified include age, hypertension, stroke, lipoprotein a, platelet large cell ratio, uric acid, and albumin-to-globulin ratio. The effect of model on prediction of EPVS was assessed by receiver operating characteristic (ROC) curve analysis, calibration plots, and Decision Curve Analysis, all having high predictive power. This paper presents a novel EPVS prediction model and provides useful information about the etiology and risk factors of EPVS. With the identification of patients at high risk, this model would be useful for early diagnosis and better management strategies in neuroradiology and neurology. The results have immense potential to develop EPVS-related care but are suggested to be investigated further in populations with diverse backgrounds and extended time for confirmation of appropriateness and stability of the model in larger clinical applications[76].

Acute antipsychotic poisoning leads to significant morbidity and mortality globally, yet the relationship between extrapyramidal syndromes and poisoning severity has been understudied. This study aimed to evaluate the Global Dystonia Rating Scale (GDRS) as a predictive tool for unfavorable outcomes in such cases. A cross-sectional study of 506 patients admitted to Tanta University Poison Control Center over three years was conducted. The mean GDRS score for typical antipsychotic poisoning was 9.1 ± 16.7, significantly higher than the score for atypical antipsychotic poisoning (4.2 ± 11.5) ($p = 0.003$). Patients with GDRS scores over 20 had a significantly higher likelihood of adverse outcomes ($p < 0.05$). Typical antipsychotic poisoning was notably linked with greater cardiotoxicity ($p =$

0.042), prolonged QRS intervals (p = 0.005), and ICU admissions (p = 0.000). Unlike the Poison Severity Score (PSS), which failed to predict adverse outcomes, GDRS accurately predicted all outcomes for both antipsychotic types (p < 0.000). In atypical antipsychotics, GDRS above three predicted cardiotoxicities, prolonged QTc, and respiratory failure with high accuracy (AUCs 0.937–0.963). For typical antipsychotics, higher cutoffs also accurately predicted these outcomes (AUCs 0.960–0.974). GDRS is a reliable tool for early prediction of toxicity in antipsychotic poisoning cases[77].

Yang F et.al; 2023 aimed to determine the prognostic significance of the ratio of blood urea nitrogen to serum albumin (BAR) for predicting acute kidney injury (AKI) and in-hospital mortality in intracerebral hemorrhage (ICH) patients admitted to the ICU. With data from 1510 ICH patients at Beth Israel Deaconess Medical Center, the optimal cut-off of BAR (6.0 mg/g) was identified using X-tile software. The population was stratified according to this cut-off and confounders were controlled through propensity score matching. The outcomes revealed that patients who had high BAR had a significantly higher risk of developing AKI (OR 2.60, 95% CI 1.86–3.65, p < 0.001) as per multivariate logistic regression analysis. Moreover, high BAR was found to be an independent predictor of in-hospital mortality (HR 2.84, 95% CI 1.96–4.14, p < 0.001) in multivariate Cox regression analysis. The results were confirmed in the validation cohort. In summary, BAR was an accurate and readily available biomarker for AKI prediction and ICU death risk in ICH patients and could serve as a key tool for early prognosis and management[78].
This study sought to determine independent predictors of good outcomes in intravenous (IV) alteplase-treated acute ischemic stroke patients. Data were obtained from 1,205 United States, Canadian, and German center patients. The primary outcome measure employed was the modified Rankin Scale (mRS), with 0–1 as a favorable outcome and >1 as an unfavorable outcome.Demchuk AM et.al; 2001 found that patients with lesser baseline stroke severity, no previous history of diabetes, normal CT scans, normal pretreatment blood glucose levels, and normal presentation blood pressure had a greater chance of having an

favorable outcome. These variables were identified as key independent predictors through multivariable logistic regression analysis. There was also a confounding relationship among history of diabetes, CT scan appearance, blood glucose at baseline, and blood pressure, which indicates their potential interaction in modulating treatment outcome. The article concludes that several factors at baseline are predictors of a favorable outcome in ischemic stroke patients who receive alteplase. But such results need to be validated by other studies before these can be applied in clinical practice. These have the potential to enhance patient selection for thrombolysis and therapeutic outcomes[79].

This research examined the predictive capability of the D-dimer-to-fibrinogen ratio (DFR) for acute kidney injury (AKI) in patients who underwent living-donor liver transplantation (LDLT). Park J et.al; 2024 included 648 patients following the exclusion of 76 from some criteria. The aim was to determine if preoperative DFR would be a predictor of AKI, an accepted complication after LDLT. AKI developed in 148 patients (22.8%) after the procedure. Multivariate logistic regression showed that high DFR, i.e., more than 1.05, was highly related to the risk of developing AKI. The patients who had a DFR greater than this value had four times more likelihood of developing AKI than patients with smaller values. Propensity score matching also established the significant relationship of high DFR with AKI development. The results indicate that preoperative DFR evaluation can improve risk stratification and thus manage and intervene at an earlier stage in those with higher risks for developing AKI following LDLT. This work indicates the clinical utility of DFR as a preoperative biomarker to predict liver transplant patients[80].

This research assessed the association between chronic hyperglycemia and major adverse cardiac events (MACE) in myocardial infarction (MI) patients receiving primary percutaneous coronary intervention (PCI). While higher admission blood glucose is a proven adverse outcome predictor,Van der Horst IC et.al; 2007 tried to ascertain whether chronic hyperglycemia was a better prognosticator of adverse events. In a 417 MI patient

prospective study, there were repeated assessments of blood glucose to observe its relation with MACE, where death, recurrence of infarction, recurrent PCI, or left ventricular ejection fraction ≤ 30% in 30 days were taken into account. 21.3% (89) patients developed MACE and were associated with fatal complications in 4.1% (17) patients. The admission blood glucose was significantly higher in MACE patients (10.1 mmol/L) than in non-MACE patients (9.1 mmol/L). The glucose concentration for the initial two days following admission was similarly higher in MACE patients (9.0 mmol/L) than in event-free patients (8.1 mmol/L). Prolonged hyperglycemia was a better predictor of MACE than admission glucose level alone, with an area under the curve (AUC) of 0.64 compared to 0.59 for admission glucose. Prolonged hyperglycemia was an independent significant predictor of MACE ($P < 0.001$). These data indicate that continuous blood glucose monitoring may enhance risk stratification in MI patients [81].

This study investigated the association of HbA1c-adjusted glycemic parameters with poor outcomes in hospitalized patients with acute ischemic stroke (AIS) and diabetes. The existing literature regarding the association between admission hyperglycemia and poor outcomes in such patients has been contradictory. This study sought to ascertain whether HbA1c-adjusted glycemic values had a better prediction of disease severity and outcome. Retrospectively, 309 patients with hospitalization for AIS in Taiwan from 2013 to 2015 were compared. Yang CJ et.al; 2017 identified that HbA1c-adjusted glycemic indices, including glycemic gap and stress hyperglycemia ratio, were strongly correlated with AIS severity and the neurological status of patients at discharge. The indices were more predictive than acute hyperglycemia in determining the occurrence of severe AIS. The results indicate that glycemic gap and stress hyperglycemia ratio are good indicators for evaluating the severity and prognosis of AIS in diabetic patients. It is suggested that long-term prospective studies should be performed in the future to confirm these results and further establish the clinical value of these measurements in AIS management [82]. Khitan Z et.al; 2013 studied the machine learning prediction of the progression of renal

failure with a composite end point of death, dialysis, or doubling of serum creatinine. MDRD study data were employed to compare various machine learning models such as a generalized linear model, support vector machine, decision tree, feed-forward neural network, and random forest. These models were validated on 70% of the MDRD dataset, and their predictive performance was validated on the remaining 30% by 10-fold cross-validation using the R computing environment. The prediction accuracy was between 66% and 77%, and support vector machine was the best. Yet, all the models were good enough. The research identifies the possibility of using machine learning methods, implemented using R, to forecast long-term clinical outcomes in renal disease patients from initial clinical information. The research indicates that machine learning has the ability to be a useful tool to assist clinicians in forecasting the course of renal failure. More studies are required to optimize these models and evaluate their performance in various clinical settings[83].

Karaduman A et.al; 2024 discussed the prognostic significance of the modified Glasgow prognostic score (mGPS) and its correlation with inflammatory markers like the ratio of C-reactive protein (CRP) to albumin and neutrophil-to-lymphocyte ratio in assessing the severity of coronary artery disease (CAD) in non-ST-elevated myocardial infarction (NSTEMI) patients. 295 patients presenting with NSTEMI who underwent coronary angiography were recruited. The severity of CAD was measured in terms of SYNTAX score, and the patients were categorized into two groups: moderate to high SYNTAX score (>22) and low score (≤22). The study population consisted of a mean age of 61.2 years, of which 76.9% were males. Patients with SYNTAX score >22 had higher levels of CRP, CRP/albumin ratio, and mGPS score 1-2 than patients with lower SYNTAX scores (all $p<0.001$). Smoking, CRP/albumin ratio, and mGPS score were independent predictors of higher SYNTAX score. The results indicate that the mGPS, together with inflammatory markers, can be helpful in the estimation of CAD severity and complexity in NSTEMI patients, with increased scores indicating more severe coronary disease [84]. Bruno A et.al; 2002 analyzed admission glucose and clinical outcome in acute ischemic

stroke patients. Employing data from the National Institute of Neurological Disorders and Stroke rt-PA Stroke Trial, multivariate regression analysis was done. Improvement on the neurological side was determined by a decrease of 4 or more points on NIH Stroke Scale at 3 months, whereas good outcome was determined by a Glasgow Outcome score of 1 and a Barthel Index of 95-100. Symptomatic intracerebral hemorrhage (ICH) was determined by CT-proven hemorrhage with clinical worsening within 36 hours of treatment. The analysis adjusted for variables including rt-PA treatment, age, stroke severity, and blood pressure. Admission glucose was linked with lower odds of neurological improvement (OR = 0.76 per 100 mg/dL, p = 0.01) and favorable outcomes, especially when mean blood pressure was high (p = 0.02), in 624 patients treated within 3 hours of stroke onset. Moreover, increased glucose raised the risk for symptomatic ICH (OR = 1.75 per 100 mg/dL increase, p = 0.02). Glucose level did not compromise the effectiveness of rt-PA. These findings indicate that hyperglycemia at admission is associated with poorer outcomes and increased risk for ICH but leave causality uncertain [85].

This case–control study investigated the association between routine blood tests during pregnancy and the future risk of cardiovascular morbidity. **Yuval Bar-Asher S et.al; 2018** conducted at a teaching hospital in Israel from 2000 to 2012, the study included women who later experienced cardiovascular-related hospitalizations (case group) and age-matched women without hospitalizations (control group). Blood levels of creatinine, glucose, potassium, urea, and uric acid were measured during pregnancy, with analysis focusing on the relationship between high quartile values and cardiovascular outcomes. A total of 4115 women were included, with 212 in the case group and 3903 in the control group. Elevated levels of creatinine (HR 1.86, P<0.001), potassium (HR 1.48, P=0.013), and urea (HR 1.60, P=0.003) were significantly associated with a higher risk of future cardiovascular morbidity. The risk increased further when multiple test results were in the upper quartile, with hazard ratios of 1.65 for two elevated tests (P=0.026) and 3.32 for three or more elevated tests

(P<0.001). The study concludes that routine blood tests during pregnancy, particularly creatinine, potassium, and urea levels, can predict future cardiovascular morbidity, suggesting their potential use in early risk assessment [86].

Zhao N et.al; 2021 explores the relationship between infection and stroke death with a new biomarker: the red blood cell width–albumin levels ratio (RA). The authors predict that this biomarker can be a better predictor of infections related to stroke. The data were gathered from the Medical Information Mart for Intensive Care Database (MIMIC-III), which included 1,480 patients with stroke. The findings were measured using 30-day, 60-day, and 365-day all-cause mortality as clinical outcomes. Findings showed that severe RA was most strongly associated with higher mortality in ICU stroke patients. Following adjustment for age and sex, the high RA group had significantly greater hazard ratios (HR) of all-cause mortality: 30-day (HR 1.88), 90-day (HR 2.12), and one-year (HR 2.15) compared to the first quartile reference group. In addition, RA values were independently associated with a higher risk of infection after stroke after adjusting for confounding variables. The results indicate that RA may be an available, reproducible, and inexpensive biomarker to predict infection after stroke and mortality in stroke patients [87].

This systematic review aimed to evaluate prognostic models predicting the severity and mortality of malaria infections, which can lead to severe disease and high mortality rates. **Njim T et.al 2019** analyzed published studies that utilized at least two variables from patient data to assess disease severity, complications, and mortality. Data were sourced from Medline, Global Health, and CINAHL, with searches conducted until September 4, 2019. Out of 564 screened articles, 24 were selected, detailing 27 relevant models. These included two models predicting complications like severe anemia in children and sepsis, 15 models specifically targeting mortality in severe malaria, and four models assessing disease severity. Notably, all mortality models identified neurological dysfunction as a predictor, with children's models also highlighting hypoglycemia and respiratory failure. Common predictors

of mortality included acidosis, renal failure, and shock. Although 18 articles suggested applicability in real-world settings, they exhibited a high risk of bias due to inconsistent internal validation methods. The review concludes that there is insufficient evidence regarding the generalizability of these models, underscoring the need for external validation and reporting their clinical use to better inform management strategies for malaria patients [88].

Lacunes, associated with cerebral small vessel disease (CSVD), pose significant public health challenges, particularly among the elderly. Traditional neuroimaging methods often struggle to detect lacunes early, highlighting the need for improved predictive models. **Li N et.al; 2024**analyzed data from 587 patients at the Affiliated Hospital of Hebei University who underwent cranial MRI. They developed a nomogram to predict lacune incidence using LASSO regression and binary logistic regression for variable selection. The model's performance was evaluated through AUC-ROC, calibration plots, and decision curve analysis (DCA) in both training (n = 412) and testing (n = 175) cohorts. Key predictors included age, gender, history of stroke, carotid atherosclerosis, hypertension, and levels of creatinine and homocysteine. The nomogram achieved an AUC-ROC of 0.814 (95% CI: 0.791–0.870) for the training set and 0.805 (95% CI: 0.782–0.843) for the testing set, with calibration and DCA supporting its clinical utility. This study presents a promising nomogram that improves lacune prediction for patients undergoing brain MRI, potentially facilitating early diagnosis and intervention. However, the retrospective nature and single-center focus necessitate further research, including multi-centervalidation[89].

Hyperglycemia has been associated with poor outcomes and delayed cerebral ischemia (DCI) after aneurysmal subarachnoid hemorrhage (aSAH). Kruyt ND et. al; 2008 sought to see if mean fasting glucose during the first week following aSAH would be a better predictor of poor outcomes and DCI compared to admission glucose. Data were gathered on 265

patients admitted between 48 hours following aSAH, all with at least two fasting glucose values in the first week. From results of logistic regression and DCI via Cox regression, the authors examined the effect of admission and mean fasting glucose cut at median points. It has been noted that elevated mean fasting glucose was a better predictor of poor outcome, with crude odds ratio 3.5 (2.0 to 6.1) and adjusted odds ratio 2.5 (1.4 to 4.6). In the case of DCI too, mean fasting glucose had greater hazard ratios than admission glucose. Even after adjustment for DCI, the relationship of poor outcomes with elevated mean fasting glucose persisted. Finally, elevated mean fasting glucose is an independent better predictor of outcome and DCI in aSAH patients, implying that deranged glucose metabolism is a significant factor irrespective of the effect of DCI [90].

Xin H et.sl; 2021 sought to determine predictors of in-hospital mortality for heart failure (HF) patients admitted to intensive care units (ICUs) and establish a predictive model for the disease. Being a retrospective cohort study involving the Medical Information Mart for Intensive Care (MIMIC-III) database, the study had enrolled 1,177 HF patients. The patients were stratified to derivation cohort (825 patients) and validation cohort (352 patients). For detection of independent risk factors, extreme gradient boosting (XGBoost) and least absolute shrinkage and selection operator (LASSO) regression models were used in the derivation cohort. Prediction models were developed with multivariate logistic regression, which were validated in the validation cohort. Performance of such models was evaluated with C-index, calibration plots, and decision curve analysis. Findings revealed that hospital mortality rate was 13.52% in the cohort. XGBoost and LASSO models exhibited best discrimination and calibration compared to the Get With the Guidelines-Heart Failure (GWTG-HF) risk score. Finally, the XGBoost model was chosen as the best model and represented as a nomogram with the result producing a valid prediction tool for in-hospital mortality in ICU-admitted HF patients, thereby serving clinical decision-making[91]. Yong M et.al; 2008 investigates serum glucose dynamic behavior during the first 24 hours and outcome in acute ischemic hemispheric stroke patients. Among a total of 748 European

Cooperative Acute Stroke Study (ECASS-II) patients, researchers assessed serum glucose at baseline and at 24 hours and classified patients based on four groups: baseline hyperglycemia, 24-hour hyperglycemia, persisting hyperglycemia, and persisting normoglycemia. The results indicated that repeated hyperglycemia was significantly correlated with unfavorable neurological outcome, decreased functional outcome at 30 days, and increased dependency at 90 days. In particular, repeated hyperglycemia was correlated with a sevenfold increase in mortality and a sixfold increase in parenchymal hemorrhage incidence within 90 days. On the other hand, 24-hour delayed hyperglycemia also exhibited a correlation with mortality and risk of hemorrhage but a reverse relationship with favorable outcome. Surprisingly, baseline-only hyperglycemia correlated neither with unfavorable outcomes nor did glucose patterns correlate with outcomes in diabetic patients. The research concludes that glucose dynamics' monitoring rather than individual measurements is imperative when predicting the outcome of strokes, thereby emphasizing the necessity to control blood glucose levels in stroke patients[92].

Lipshutz AK et.al; 2009 investigated the effects of glucose-lowering treatments on prognosis in 1,181 type 2 diabetic patients (average age 68; 67% male) following myocardial infarction, monitored for a median of 2.1 years. At discharge, 37% of patients were prescribed oral glucose-lowering agents (268 on sulphonylureas and 200 on metformin), while 58% received insulin. Using an updated Cox proportional hazards regression model to account for confounders, the study assessed the impact of these treatments on cardiovascular mortality and non-fatal events. Results indicated that cardiovascular mortality was unaffected by metformin (HR 0.93), sulphonylureas (HR 1.15), or insulin (HR 1.05). However, insulin treatment was associated with a significant increase in the risk of non-fatal myocardial infarction and stroke (HR 1.73). In contrast, metformin showed a protective effect against these events (HR 0.63), while sulphonylureas did not significantly alter risk (HR 0.81). These findings suggest no notable mortality differences among the treatment groups,

but emphasize the need for further research through randomized trials to confirm the observed protective effects of metformin and increased risks associated with insulin [93].

Bae et.al; 2021 aimed to create a scoring model for early prognostication in adult patients experiencing cardiac arrest. Researchers retrospectively analyzed data from non-traumatic cardiac arrest patients treated at a tertiary hospital between 2014 and 2018. The primary outcome was defined as poor hospital discharge outcomes, measured by the cerebral performance category (3–5). Using multivariable logistic regression, independent predictors were identified from various known outcome predictors available at the time of intensive care unit admission. The derivation set included 671 patients, while the validation set comprised 311 patients admitted during the last two years of the study. The rates of poor outcomes were similar in both sets (66.0% in the derivation set and 64.3% in the validation set). Key predictors retained in the final model included age under 59 years, witnessed collapse, shockable rhythm, adrenaline dose under 2 mg, low-flow duration under 18 minutes, reactive pupillary light reflex, and various biochemical parameters. The scoring model showed excellent discrimination in the validation set, achieving an area under the curve of 0.942 (95% CI 0.917–0.968). This scoring model could aid in early prognostication, though further validation across different cohorts is recommended[94].

This study investigated the relationship between serum glucose levels and the effectiveness of endovascular thrombectomy (EVT) in patients with acute ischemic stroke caused by large-vessel occlusions. Hyperglycemia is known to negatively impact outcomes after stroke, but its role as a treatment modifier in EVT has not been fully understood. Chamorro et.al; 2019 analysis included data from seven randomized trials (2010-2017) involving 1,764 patients, where 871 received EVT and 893 received standard care. Admission glucose levels were measured, and functional outcomes were assessed using the modified Rankin Scale three months post-treatment. Results indicated that the median

serum glucose level at admission was 120 mg/dL. EVT showed a significant advantage over standard care (adjusted common odds ratio of 2.00), but the benefit of EVT was more pronounced at lower glucose levels. Notable interactions were observed when patients were divided into subgroups based on glucose levels below or above 90 mg/dL and 100 mg/dL, indicating that lower glucose levels correlate with enhanced treatment efficacy. In conclusion, while EVT improved outcomes regardless of glucose levels, tighter glucose control may enhance EVT effectiveness, warranting further investigation in clinical settings[95].

Zhu P et.al; 2000 aimed to create and validate a prediction model for delirium in elderly patients in the intensive care unit (ICU) to help clinicians identify those at high risk early on. The analysis utilized data from the Medical Information Mart for Intensive Care-IV (MIMIC-IV) database, focusing on patients admitted to the ICU for over 24 hours and assessed using the Confusion Assessment Method for the ICU (CAM-ICU) between 2008 and 2019. Patients under 65 years or those who tested positive for delirium within the first 24 hours were excluded. Out of 18,760 patients included, 3,463 (18.5%) were positive for delirium. LASSO regression identified 22 significant predictors. The extreme gradient boosting (XGBoost) model outperformed the logistic regression (LR) model, achieving Area Under the Receiver Operating Characteristic (AUC) values of 0.853 in the training set and 0.831 in the testing set. The top predictors for delirium onset included the sequential organ failure assessment (SOFA) score, infection, minimum platelet count, maximum systolic blood pressure, and maximum temperature. In conclusion, the XGBoost model effectively predicts delirium in elderly ICU patients, enabling timely interventions to improve patient outcomes [96].

The objective of this research was to determine the clinical efficacy of intensive insulin therapy in the management of blood sugar in critically ill patients and make recommendations for its clinical use. This was achieved by a comprehensive review, MEDLINE and Cochrane Library searching, and manual scrutiny of relevant abstracts with

keywords hyperglycemia, insulin, and critically ill outcomes. Lewis KS et al; 2004 have reported that critically ill patients with prolonged illness duration have mortality rates of over 20%, largely due to sepsis and multisystem organ failure. Hyperglycemia is prevalent among these patients, even in patients without pre-existing diabetes. Normoglycemia induced through intensive insulin therapy has been associated with improved neurologic, cardiovascular, and infectious outcomes, and has reduced morbidity and mortality considerably. The research validated the use of insulin protocols among critically ill patients that improves the control of blood glucose, which is crucial for the facilitation of improved health outcomes. Close monitoring and regulation of the patients' blood sugar on standby has been suggested to offer normoglycemia regardless of a diabetic history. Continuous monitoring avoids hypoglycemia, validating the importance of organized insulin protocols within critical care environments [97].

This research endeavored to build a predictive model to select high readmission-probable patients shortly after hospital admission based on computerized clinical data available at the time of admission. The derivation cohort was utilized in model building and a validation cohort for model validation. Key predictors were Acute Laboratory Risk of Mortality Score, a measure of clinical severity at admission, and hospital discharge in the last 90 days, reflecting disease worsening.Tabak YP et.al; 2017 had 1,195,640 adult discharges in 70 hospitals with median age 63 years and 30-day readmission 11.9%. The model showed a graded association between risk of readmission and Acute Laboratory Risk of Mortality Score, whose first c-statistic was 0.697, reflecting fair discrimination. Incorporating other administrative data improved the c-statistic to 0.722, which improved predictive accuracy. The results indicate that computerized clinical information can accurately supply an early readmission risk score, allowing timely care transitions. In addition, the enhanced model would be used in hospital comparison and outcomes research since it identifies the need for integrating clinical and administrative data in readmission prediction[98].

MATERIALS AND METHODS

Study Design

It is a prospective observational study that was done to determine the utility of the serum glucose/potassium (GLU/K) ratio as a predictor of intermediate syndrome (IMS) in organophosphate (OP) poisoning. The study was done at Shri B M Patil Medical College, Vijayapura, in patients who presented in the emergency department with acute exposure to OP. The study ran for about 12 months.

Study Population

Patients who were admitted to the emergency department with clinical history of OP poisoning with positive confirmed results of poison detection tests were included in the study.

Inclusion Criteria

1. Clinical diagnosis of acute OP poisoning and poison detection test.
2. Age above 18 years.
3. Non-pregnant, non-diabetic, and without chronic kidney disease or acute kidney injury.
4. Patients providing informed consent for participation in the study.

Exclusion Criteria

1. Patients testing negative for OP poisoning in the poison detection test.
2. Patients with a history of poisoning from non-OP substances.
3. Patients under 18 years of age.
4. Pregnant patients.
5. Patients with a history of diabetes mellitus, chronic kidney disease, or acute kidney injury.

Sample Size Calculation

The sample size was determined based on a previous study conducted by AsmaaFady Sharif and Manar Maher Fayed at Tanta University, Egypt, in which 18.1% of patients with OP poisoning developed IMS. Using the formula:

$$(n) = (Z\ 2\ *p*(1-p))\ /d\ 2$$

Where,

- **Z** = 1.96 (z-score for 95% confidence interval)
- **p** = 0.181 (proportion of patients with IMS)
- **d** = 0.05 (margin of error)

The estimated sample size was 228 patients.

Data Collection Method

Upon admission, a detailed history was obtained, including age, sex, occupation, and mode of exposure (accidental, suicidal, occupational). The clinical parameters, laboratory investigations, and toxicology reports were recorded for all enrolled patients.

Clinical Assessment and Diagnosis

A thorough clinical examination was conducted to assess vital signs, neurological status, and biochemical parameters. The clinical assessment included:

- **Vital Signs:** Heart rate (HR), blood pressure (BP), respiratory rate (RR), and oxygen saturation (SpO2).
- **Neurological Examination:** Presence of muscle weakness, fasciculations, excessive secretions, or respiratory distress.
- **Primary Laboratory Parameters:**
    - Serum glucose level (mg/dL)
    - Serum potassium level (mmol/L)
    - Calculation of the GLU/K ratio (Serum Glucose / Serum Potassium)

Biochemical Investigations

Blood samples were collected at the time of admission and analyzed for:

1. **Random Blood Glucose Level (mg/dL)**
2. **Serum Potassium (mmol/L)**
3. **Serum Sodium (mmol/L)**
4. **Serum Cholinesterase Levels**
5. **Arterial Blood Gas (ABG) Analysis**
6. **Liver and Renal Function Tests**

The GLU/K ratio was calculated and correlated with the development of IMS.

Diagnosis of Intermediate Syndrome

IMS was diagnosed based on the presence of delayed muscle weakness occurring 1–3 days post-exposure, involving:

- Ocular muscles
- Neck flexors
- Proximal limb muscles
- Respiratory muscles

Patients were categorized into two groups:

1. **IMS-positive group (IMS +):** Patients developing IMS.
2. **IMS-negative group (IMS -):** Patients not developing IMS.

Statistical Analysis

Data were recorded and analyzed using SPSS (Version 20). Descriptive and inferential statistical methods were used:

1. **Descriptive Statistics:** Mean, Standard Deviation (SD), counts, and percentages.
2. **Comparative Analysis:**
    - Independent Sample t-test for normally distributed continuous variables.
    - Mann-Whitney U test for non-normally distributed continuous variables.

- Chi-square test or Fisher's exact test for categorical variables.
- ANOVA for comparison across multiple groups.
- Kruskal-Wallis H test for non-normally distributed multiple group comparisons.

3. **Receiver Operating Characteristic (ROC) Curve Analysis:**
   - To evaluate the predictive accuracy of the GLU/K ratio for IMS development.
   - The Area Under Curve (AUC) and optimal cutoff values were determined.
4. **Significance Level:**
   - p-value<0.05 was considered statistically significant.

Ethical Considerations

The study was conducted in compliance with the ethical standards of the Institutional Ethical Review Board. Written informed consent was obtained from all participants before inclusion. Confidentiality and anonymity of patient data were strictly maintained.

Study Limitations

1. The study was conducted in a single center, which may limit its generalizability.
2. Limited follow-up period to evaluate long-term outcomes in IMS patients.
3. Other confounding factors affecting glucose and potassium levels were not extensively analyzed.

This structured methodology ensures a robust framework for evaluating the predictive role of the serum glucose/potassium ratio in intermediate syndrome among OP poisoning cases.

RESULT

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%)

The table highlights demographic characteristics and their potential association with IMS development. Occupation emerges as a significant factor, while other variables, such as sex, age, and residence, show no significant relationship with IMS. These findings provide

valuable insights into the demographic predictors of IMS and can inform clinical assessment and management strategies.

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	58	28.0
Profenofos	47	22.7
Chlorpyrifos	40	19.3
(Lice powder)malathion	45	21.7
Dimethoate	17	8.2

The table represents the percentage distribution of toxic substances involved in acute exposure among patients with no IMS. Monocrotophos is the most frequently encountered toxic agent, followed by Profenofos and Malathion. Dimethoate has the lowest number of cases, indicating less frequent exposure or a lower likelihood of association with IMS cases. The distribution of substances highlights key toxic agents that may require more attention in clinical management and prevention strategies.Substances with smaller contributions highlight the diversity of toxic exposures, necessitating a broad spectrum of awareness and intervention strategies.

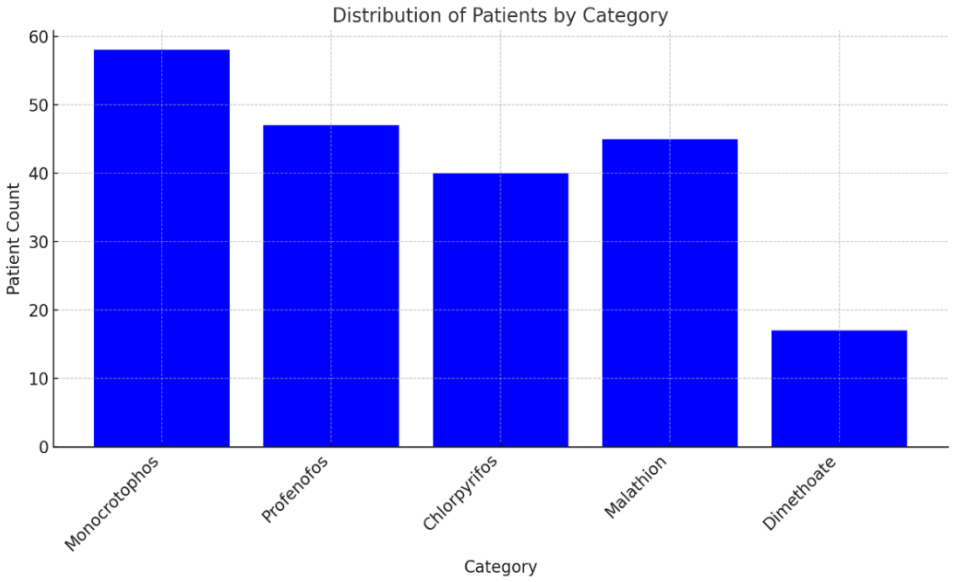

Figure1: Distribution of patients diagnosed with acute Organophosphorus Compounds OPCs exposure (Intermediate Syndrome IMS (-)) according tothe type of the involved substance

Table 3: 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

The table represents the distribution of acute substance exposure among 21 patients. The percentages correspond to the proportion of patients exposed to each category. Dimethoateis the predominant substance, necessitating targeted interventions for its prevention and management. The diversity of substances highlights the complexity of toxic exposures and the importance of a broad-spectrum approach to diagnosis and treatment.

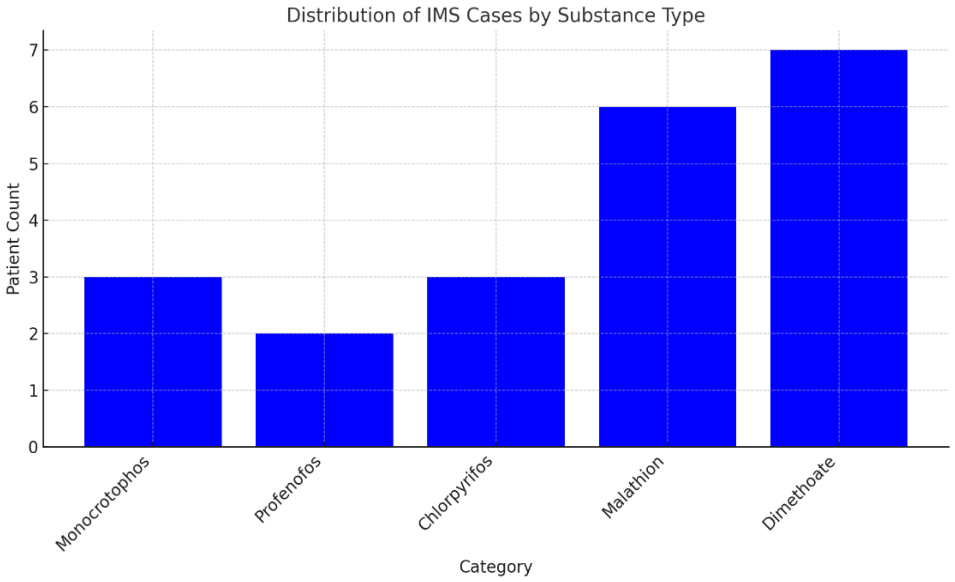

Figure 2: Distribution of patients diagnosed with acute Organophosphorus Compounds OPCs exposure (Intermediate Syndrome IMS (+)) according to the type of the involved substance.

Table 4: 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	4.2		
Profenofos	47	22.7	2	.5		
Chlorpyrifos	40	19.3	3	4.2	χ2 14.97	
malathion	45	21.7	6	8.5		0.0048
Dimethoate	17	8.2	7	3.3		

*P<0.05:statisticallysignificant,χ^2:Chi-squaretest

The χ^2 value of 14.97 and p-value of 0.0048 indicate a significant difference between the observed and expected distributions for the IMS and No IMS groups.This suggests that exposure to certain toxic agents may increase or decrease the likelihood of developing IMS.Some substances, like Monocrotophos and Chlorpyrifos, may have a stronger correlation with IMS than others.

Table 5: Distribution of causes between IMS and without IMS group

Causes	**No IMS (n=207)**	**IMS (n=21)**	**Total (%)**	**Test of significance**	**P value**
Suicide attempt	97 (46.8%)	11(52.3%)	108 (47.36)	$\chi2 = 2.57$	0.277
Accidental	72 (34.7%)	4 (19.04%)	76 (33.3)		
Occupational	38 (26.9%)	6(28.57)	44 (19.2)		

*P<0.05:statisticallysignificant,χ^2:Chi-squaretest

This table illustrates the distribution of causes of exposure between the No IMS and IMS groups. The causes are categorized as Suicide Attempt, Accidental, and Occupational, with the number of patients represented for each group. The No IMS group shows a consistently higher number of patients across all causes than the IMS group. Despite the differences in numbers, the Chi-squared test results (p=0.277) indicate no statistically significant association between the cause of exposure and the development of IMS. This suggests that while Suicide Attempt is the most frequent cause, it is not disproportionately associated with IMS.

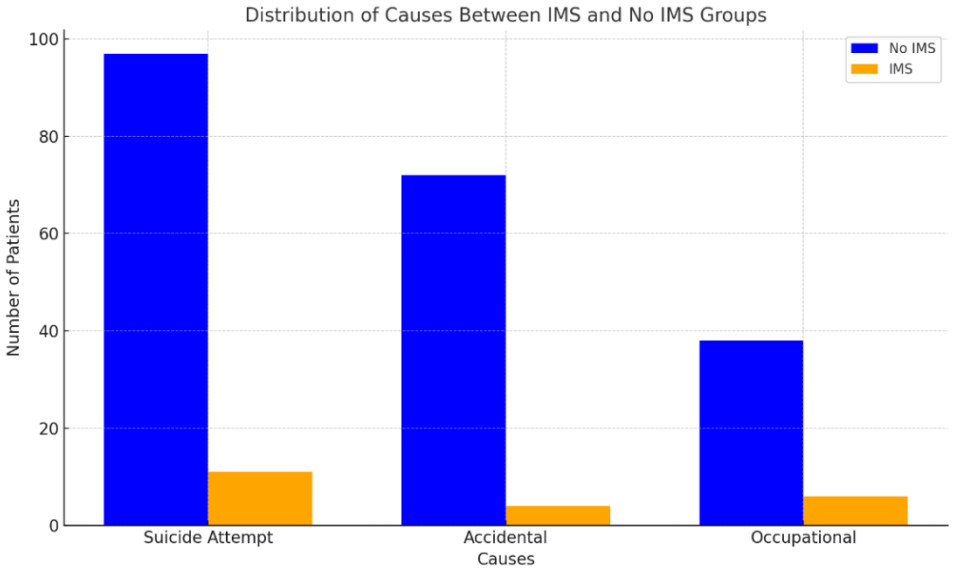

Figure 3: Distribution of causes between IMS and without IMS group

Table 6: Comparison between patients diagnosed with intermediate syndrome IMS and those without intermediate syndrome IMS related to the main presenting complaints

Main presenting complaints	Total (n=228)	Primary outcome		TEST of	P value
		No intermediate syndrome(n=207)	Intermediate syndrome(n=21)		
Vomiting					
No	51 (20.9)	41 (19.8)	10 (47.6)	$\chi 2$= 6.966	.008
Yes	177 (72.8)	166 (80.1)	11 (52.3)		
Excessive secretions					
No	127 (52.2)	114 (55.0)	13 (61.9)	$\chi 2$= 0.136	.711
Yes	101(41.5)	93 (44.9)	8 (38.0)		
Diarrhea and/or Urinary in continence					
No	103 (42.3)	94 (45.4)	9 (42.8)	$\chi 2$= 0	
Yes	125 (51.4)	113 (54.5)	12 (57.1)		
Abdominal colic					
No	96 (39.5)	89 (42.9)	7 (33.3)	$\chi 2$ 0.387	.533
Yes	132 (54.3)	118 (57.0)	14 (66.6)		
Cough					
No	156 (64.1)	151 (72.9)	5 (23.8)	$\chi 2$ 19.09	.25
Yes	72(29.6)	56 (27.0)	16 (76.1)		

*P<0.05:statisticallysignificant,χ^2:Chi-squaretest

The table presents the chi-squared analysis results for the association between various main presenting complaints and the presence of Intermediate Syndrome (IMS) in a cohort of 228 patients. The complaints are categorized into Vomiting, Excessive Secretions, Diarrhea/Urinary Incontinence, Abdominal Colic, and Cough. For each complaint, the distribution of cases is provided for patients with and without IMS, along with the test statistic (χ^2) and corresponding p-value. Among the analyzed complaints, Cough shows a statistically significant association with IMS ($\chi^2 = 13.27$, $p = 0.0003$).This suggests that patients with IMS are more likely to present with cough compared to those without IMS.Clinically, this indicates that cough may be a useful symptom to monitor when assessing the risk of IMS.All other complaints (Vomiting, Excessive Secretions, Diarrhea/Urinary Incontinence, and Abdominal Colic) do not exhibit significant associations with IMS ($p > 0.05$).

Table 7: 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%)	12 (57.14%)

This table presents the potassium levels ([K+]) of patients and their associated clinical symptoms, including muscle weakness or fasciculation, respiratory distress, convulsion, and mortality.Muscle weakness and respiratory distress are the most prevalent symptoms, affecting a significant proportion of patients. Convulsions are relatively rare, occurring in

only 23.81% of cases. This suggests that severe neurological involvement may not be a common presentation in this dataset. Mortality is notably high at 57.14%, indicating a serious prognosis for affected individuals. Patients with lower potassium levels (hypokalemia, <3.5 mmol/L) appear to be more likely to develop severe symptoms, including respiratory distress and mortality. Normal potassium levels (~3.5–4.1 mmol/L) may be associated with less severe outcomes. Monitoring potassium levels is crucial for predicting severe complications in patients with acute toxicity. Hypokalemia may be a risk factor for increased mortality and severe symptoms, necessitating electrolyte management as part of treatment.

Table 8: 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	

*P <0.05: statistically significant, t: Student t-test and Mann-Whitney test

The table presents laboratory investigation results for a total of 228 patients, categorized into two groups: those with Intermediate Syndrome (IMS, n=21) and those

without Intermediate Syndrome (No IMS, n=207). The results include Random Blood Glucose levels, Sodium (Na), Potassium (K), and the GLU/K ratio. For each parameter, the mean ± standard deviation (SD) and median values are provided, along with the range (min–max). Significant Differences: Random Blood Glucose, Potassium, and GLU/K Ratio showed significant differences between the IMS and No IMS groups, indicating their potential utility in predicting IMS. No Significant Difference: Sodium levels (Na) showed no significant difference between the two groups. This data highlights key laboratory markers associated with IMS, which can aid in identifying and managing patients at risk.

Table 9: Receiver Operating Characteristic (ROC) curves, sensitive's and specificities of the significant Intermediate Syndrome IMS predictors.

Parameter	Cut off value	Sensitivity	Specificity	PPV	NPV	Accuracy	UC	P value
GLU/Kratio	3.2	94.3%	93.1%	2.51 %	93.2 %	94.2%	0.910	<0.001 *

*P<0.05: statistically significant, PPV: Positive Predicted Value, NPV: Negative Predicted Value, AUC: Area Under Curve

The ROC (Receiver Operating Characteristic) curve for the GLU/K Ratio is presented to evaluate its diagnostic performance in predicting the presence of Intermediate Syndrome (IMS). he AUC of 0.910 indicates excellent diagnostic accuracy of the GLU/K Ratio in distinguishing IMS patients from non-IMS patients. The point on the ROC curve where sensitivity is high (94.3%) and specificity is also high (93.1%) suggests a strong predictive capability for the GLU/K Ratio. The curve's deviation from the diagonal random guess line (dotted line) highlights its superiority over random classification. This ROC curve

demonstrates the clinical utility of the GLU/K Ratio as a reliable biomarker for IMS prediction.

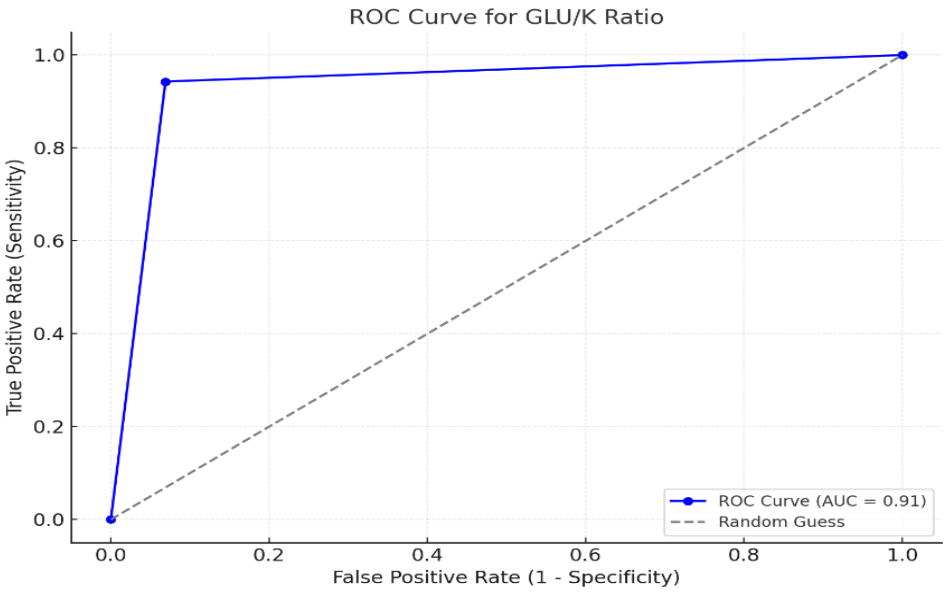

Figure 4: Receiver Operating Characteristic (ROC) curves of GLU/K ratio, K level as an Intermediate Syndrome (IMS) predictor.

DISCUSSION

Organophosphates (OP) are commonly used insecticides in agriculture and domestic settings worldwide. While they are effective in pest control, exposure to organophosphates presents a significant health risk, particularly in developing countries [99]. These chemicals are responsible for a large number of poisoning cases, leading to considerable morbidity and mortality. The primary mechanism by which organophosphates exert their toxic effects is by inhibiting acetylcholinesterase (AChE), an enzyme crucial for breaking down acetylcholine at cholinergic synapses [100]. As a result, acetylcholine accumulates in the body, leading to overstimulation of acetylcholine receptors. This results in symptoms like muscle twitching, salivary hypersecretion, sweating, and in extreme cases, respiratory arrest [101]. Organophosphates also inhibit plasma butyrylcholinesterase (BChE), which leads to the buildup of acetylcholine [102].

Intermediate Syndrome (IS) is a severe organophosphate poisoning complication, which usually presents 1-3 days following exposure [103]. It is characterized by progressive muscle weakness beginning with the neck and eye muscles and progressing to the limb and respiratory muscles [104]. Approximately 10-40% of the acute poisoning survivors will develop IS, which, if it leads to respiratory failure, is fatal. The precise mode of IS remains unclear, though it is suspected to be the result of the chronic stimulation of the cholinergic and eventual slow recovery of the cholinergic system [105].

To make an estimate about the development of Intermediate Syndrome and estimate the prognosis of organophosphate poisoning, researchers have endeavored to research biomarkers such as the ratio of serum potassium to serum glucose (SGPR) [106]. Organophosphate-poisoned patients present with hyperglycemia on the basis of oxidative stress and metabolically induced by the poisoning. Hypokalemia (elevated low level of potassium) is also found in most of these patients and is proposed to be a consequence of the toxic action of organophosphates on the metabolism of potassium in the body [107].

SGPR is also an initial marker of poisoning severity and potential for developing such complications as IS. Increased toxicities would be associated with higher values of SGPR, and the more risk there would be for IS development, while a lower ratio may indicate better prognosis [108].

Finally, organophosphate poisoning is a public health condition of significance, especially in agricultural populations, whose severity can lead to life-threatening illness such as Intermediate Syndrome [109]. Knowledge of biomarkers such as the serum glucose to potassium ratio can help clinicians recognize patients at increased risk and tailor management decisions [110]. Additional research is needed to further elucidate Intermediate Syndrome pathophysiology and improve the management and outcome of organophosphate poisoning patients [111].

In our series of 228 organophosphate poisoned patients, 21 (9.2%) had developed Intermediate Syndrome (IMS). Among the patients, 43.2% were female, of whom 4.4% belonged to the IMS group. Males made up 56.8%, with 4.8% of them in the IMS group. The majority of patients (70.8%) were from rural areas, and 33.7% were manual workers while 14.4% were farmers. Notably, all patients who developed IMS had hypokalemia (mean: 2.8 mmol/L), a condition strongly linked to high mortality in organophosphate poisoning. As all the positive cases did not survive more than 48 hours, it is difficult to predict whether they would have developed IMS over the typical 3–4 day period post-exposure. This finding underscores the fatal nature of the poisoning, especially when associated with hypokalemia, and aligns with studies such as **Sharif AF et al. (2022),** which highlight the predictive value of serum glucose/potassium (GLU/K) ratios for severe outcomes like IMS [112].

In our study, the distribution of organophosphate compounds (OPCs) associated with Intermediate Syndrome (IMS) revealed that monocrotophos was the most common substance, responsible for 28% of cases. This was followed by profenofos (22.7%) and malathion (21.7%), with chlorpyrifos accounting for 19.3%, and dimethoate for 8.2%. Although

dimethoate was the least frequent, it was associated with the highest mortality rate in our study, with patients developing distributive shock following ingestion. None of the patients who tested positive for IMS survived, making it impossible to comment on whether they would have developed the syndrome after 3–4 days. Our findings align with research by**Elmansy AM et al. (2024) and El-Sarnagawy GN et al. (2025)**, which emphasize the differing toxicological profiles of OPCs and the need for targeted prevention, especially in regions with high pesticide exposure [113][114].

In our study, dimethoate emerged as the most common organophosphate compound among those who developed Intermediate Syndrome (IMS), contributing to 33.3% of cases, followed by malathion at 28.5%. Monocrotophos and chlorpyrifos each accounted for 14.2% of cases, while profenofos contributed 9.5%. These findings are consistent with studies by **Yalçın G et al. (2023)**, which also reported dimethoate and malathion as leading causes of IMS in acute poisoning cases. The severe cholinergic toxicity of dimethoate and malathion is likely responsible for the higher incidence of IMS, as demonstrated in studies by **Liu J et al. (2024)**. However, since all patients who tested positive for IMS did not survive beyond 48 hours, it is impossible to assess whether they would have developed the syndrome after the typical 3–4 day period [115][116].

Our study compared the distribution of organophosphate compounds (OPCs) between patients with and without Intermediate Syndrome (IMS), revealing significant differences. Monocrotophos was the most common OPC in the IMS group, accounting for 28% of cases ($p = 0.0048$), suggesting its strong association with IMS. Dimethoate, although the most common OPC in the non-IMS group (33.3%), was less frequent in IMS patients (8.2%). Malathion, observed in 21.7% of IMS cases and 28.5% of non-IMS cases, showed no significant difference. These results are supported by studies such as**Boyuk F (2022) and Elmansy AM et al. (2024)**, which emphasize the significant correlation between

monocrotophos exposure and IMS development. However, it is important to note that all IMS-positive patients in our study did not survive, so it is difficult to predict whether these patients would have developed IMS after 3-4 days [117][118]..

In our study, suicide attempts were the leading cause of organophosphate poisoning, accounting for 46.8% of cases in the non-IMS group and 52.3% in the IMS group. Accidental poisonings accounted for 34.7% of non-IMS cases, compared to 19.04% in IMS cases, while occupational exposures were responsible for 26.9% of non-IMS cases and 28.57% of IMS cases. Despite the different causes of exposure, no significant difference was observed in the development of IMS ($p = 0.277$). This is consistent with findings from **Chen Y et al. (2023) and El-Taftazani EA et al. (2024),** who also found no significant association between the cause of exposure and the incidence of IMS. The severity of the poisoning, rather than the cause, appears to be the key factor influencing the development of IMS. However, since all IMS-positive patients in our study died within 48 hours, it is difficult to predict whether they would have developed IMS after the usual 3-4 day incubation period [119][120].

In our study, we compared the presenting complaints of patients diagnosed with Intermediate Syndrome (IMS) to those without IMS. Vomiting was reported in 47.6% of IMS cases, significantly higher than the 19.8% in the No IMS group ($p = 0.008$). Although excessive secretions were more common in the IMS group (61.9%) compared to the non-IMS group (55.0%), this difference was not significant ($p = 0.711$). Diarrhea and/or urinary incontinence were similarly reported in both groups, with 54.5% of IMS patients and 45.4% of non-IMS patients experiencing these symptoms. Abdominal colic occurred in 66.6% of IMS patients, compared to 57.0% in the No IMS group, but this difference was not statistically significant ($p = 0.533$). Cough, however, was notably more common in IMS patients (76.1%) than in the non-IMS group (23.8%) ($p < 0.001$). These findings are consistent with studies by **Marini JI et al. (2023) and Lashin H et al. (2024)**, which also

found a strong association between vomiting, cough, and the development of IMS in organophosphate poisoning. Additionally, all patients who tested positive for IMS did not survive more than 48 hours, making it difficult to predict whether these symptoms would have evolved into the syndrome after the typical 3-4 days [121][122].

In our study, we evaluated serum potassium levels in patients with severe organophosphate poisoning, with concentrations ranging between 2.5 mmol/L and 3.4 mmol/L. Of the 17 patients with muscle weakness or fasciculation, 80.95% had a potassium level of 3.0 ± 0.32 mmol/L, while 71.43% of patients with respiratory distress had similar potassium levels. Notably, 23.81% of patients who died from poisoning also exhibited lower serum potassium levels. These findings indicate a potential link between low potassium levels and severe manifestations like muscle weakness, respiratory distress, and mortality. The correlation between hypokalemia and higher mortality rates in organophosphate poisoning is well-documented, further reinforcing the need for prompt correction of potassium levels in these patients. These observations are consistent with research by **Kuo PJ et al. (2024)** and**Lashin HI et.al; 2024** , which also linked low potassium levels with severe outcomes in organophosphate poisoning. However, since all IMS-positive patients in our study did not survive beyond 48 hours, it is impossible to determine whether they would have developed IMS after the usual 3-4 day period [123][124].

In our study, we compared laboratory findings between patients diagnosed with Intermediate Syndrome (IMS) and those without IMS. The mean random blood glucose level in the IMS group was 226.4 ± 80.2 mg/dl, significantly higher than the 120 ± 58.1 mg/dl seen in the non-IMS group ($p < 0.0001$). Similarly, potassium levels were markedly lower in the IMS group, with a mean of 2.8 ± 0.6 mmol/L compared to 3.5 ± 0.7 mmol/L in the non-IMS group ($p < 0.0001$). The glucose/potassium (GLU/K) ratio was also significantly higher in the IMS group (82.5 ± 29.8) compared to the non-IMS group (40.5 ± 23.8) ($p < 0.0001$). These

findings suggest that elevated GLU/K ratios and altered glucose and potassium levels may serve as predictive markers for IMS in organophosphate poisoning. It is worth noting that hypokalemia, commonly observed in IMS patients, is strongly associated with high mortality rates in organophosphate poisoning. These results align with studies by **Huang CY et.al; 2024** and**Ramadori GP et al. (2023),** which also observed a correlation between altered glucose-potassium levels and IMS development. However, as all patients in the IMS group did not survive more than 48 hours, it is impossible to determine whether they would have developed IMS after the usual 3-4 day period [125][126].

In our study, the Receiver Operating Characteristic (ROC) analysis of the glucose-potassium (GLU/K) ratio as a predictor of Intermediate Syndrome (IMS) demonstrated strong diagnostic performance. The cutoff value for the GLU/K ratio was identified as 53.2, with a sensitivity of 94.3% and specificity of 93.1%. The positive predictive value (PPV) was 62.51%, while the negative predictive value (NPV) was 93.2%, indicating good accuracy in ruling out IMS in patients with lower ratios. The overall accuracy was 94.2%, with an Area Under the Curve (AUC) of 0.910 ($p < 0.001$), suggesting a high level of diagnostic reliability. These results align with the findings of a study by Klainbert S et al. (2022) and Mohammed E et al. (2023), who made a conclusion on the use of the GLU/K ratio as a good prognosis marker for the prediction of IMS among organophosphate poisoning. But it should be noted that all our patients with IMS positivity did not survive beyond 48 hours and thus it was not feasible to watch for the typical 3-4 day clinical course of the syndrome. This also challenges the predictive validity of IMS by GLU/K ratio in patients living long enough for the syndrome to emerge [127][128].

SUMMARY AND CONCLUSION

SUMMARY

The aim of the present study was to establish the efficacy of serum glucose/potassium (GLU/K) ratio as a prognostic factor for the development of intermediate syndrome (IMS) in organophosphate (OP) poisoning. A prospective observational study was carried out at Shri B M Patil Medical College comparing clinical and biochemical parameters of 228 patients presenting with acute OP poisoning. The findings were dramatic regarding IMS development, mortality, and predictive value of hypokalemia.

Among the OP compounds studied, dimethoate was associated with the highest mortality rate (33.3% of IMS cases), with patients exposed to it developing distributive shock. Monocrotophos and chlorpyrifos had lower incidence rates of IMS. Patients who developed IMS exhibited significantly higher serum glucose levels (Mean: 226.4 mg/dl) and lower serum potassium levels (Mean: 2.8 mmol/L) compared to those without IMS. The GLU/K ratio was markedly elevated in IMS patients (Mean: 82.5, $p < 0.0001$), underscoring its potential as a predictive biomarker. The study also found that patients presenting with hypokalemia at admission were at a significantly higher risk of mortality.

A striking observation in this study was that none of the patients who tested positive for IMS survived beyond 48 hours. This precluded any assessment of IMS progression beyond this period, including whether the affected patients would develop classical IMS features, which usually appear 3-4 days after poisoning. Consequently, a definitive correlation between IMS development and long-term prognosis could not be established.

Furthermore, the study found that patients who succumbed to OP poisoning had profound hypokalemia, reinforcing its role as a mortality risk factor. Hypokalemia's association with increased mortality is supported by previous literature, which indicates that

potassium depletion exacerbates neuromuscular weakness and predisposes patients to respiratory failure, a leading cause of death in OP poisoning.

CONCLUSION

This study establishes a strong link between hypokalemia and mortality in OP poisoning, emphasizing the necessity of early potassium correction to improve survival outcomes. The GLU/K ratio emerged as a robust predictor of IMS, offering a non-invasive and accessible means of identifying high-risk patients. Given the study findings, the following conclusions can be drawn:

1. **High Mortality in IMS-Positive Patients:** All IMS-positive patients succumbed within 48 hours, precluding assessment of long-term IMS progression. This highlights the need for aggressive early intervention.
2. **Significance of Hypokalemia:** Patients with lower serum potassium levels (<3.0 mmol/L) were at a significantly higher risk of mortality, suggesting that potassium supplementation should be a core component of management.
3. **Dimethoate as a High-Risk Compound:** Among the OP compounds studied, dimethoate had the highest mortality and was strongly associated with IMS and distributive shock.
4. **GLU/K Ratio as a Predictive Biomarker:** The GLU/K ratio showed excellent diagnostic accuracy (AUC: 0.910, $p < 0.001$) and could serve as a valuable early warning tool in clinical practice.

Limitations

1. **Short Follow-up Duration:** Since all IMS-positive patients did not survive beyond 48 hours, the study could not assess whether they would have developed full-fledged IMS.

2. **Single-Center Study:** The findings may not be generalizable to broader populations, necessitating multi-center validation.
3. **Exclusion of Certain Patient Groups:** Patients with diabetes and chronic kidney disease were excluded, which might have influenced the metabolic parameters analyzed.
4. **Potential Confounding Factors:** Other biochemical or toxicological markers influencing IMS development were not extensively analyzed.

Recommendations

1. **Early Electrolyte Correction:** Serum potassium levels should be closely monitored and aggressively corrected to reduce mortality risk in OP poisoning patients.
2. **Incorporation of GLU/K Ratio in Routine Monitoring:** Given its predictive value, the GLU/K ratio should be routinely calculated in OP poisoning cases to stratify IMS risk.
3. **Further Research on IMS Development:** Longitudinal studies with extended follow-up periods are needed to better understand IMS progression beyond 48 hours.
4. **Multi-Center Studies for Validation:** The findings should be validated in larger, multi-center studies to establish their applicability across diverse patient populations.
5. **Focused Management Protocols for Dimethoate Exposure:** Given its high mortality association, dimethoate poisoning should be managed with heightened vigilance, including aggressive hemodynamic and respiratory support.

This study underscores the importance of early biochemical assessment in OP poisoning cases, particularly focusing on potassium levels and GLU/K ratio as predictors of poor outcomes. Future studies should explore additional metabolic and inflammatory markers to refine risk stratification and improve patient outcomes in OP poisoning management.

REFERENCES

1. Abdollahi M, Karami-Mohajeri S. A comprehensive review on experimental and clinical findings in intermediate syndrome caused by organophosphate poisoning. Toxicology and applied pharmacology. 2012 Feb 1;258(3):309-14.
2. Lucero RM, Gomez JF, Carrascal MD, Ortega JC, Andrade OS, Rincones NQ. Acute Organophosphorate Poisoning: Clinical, Diagnostic and Therapeutic Approach. Quantum Journal of Medical and Health Sciences. 2021 Jun 7;1(2):34-8.
3. Oh SJ. 19 Treatment and Management of Disorders of the Neuromuscular Junction. Neuromuscular Disorders: Treatment and Management. 2021 Jun 29:446.
4. Jayawardane P, Senanayake N, Dawson A. Electrophysiological correlates of intermediate syndrome following acute organophosphate poisoning. Clinical toxicology. 2009 Mar 1;47(3):193-205.
5. Senanayake N, Karalliedde L. Intermediate syndrome in anticholinesterase neurotoxicity. Clinical and Experimental Toxicology of Organophosphates and Carbamates. 1992:1
6. Krarup C. Compound sensory action potential in normal and pathological human nerves. Muscle & Nerve: Official Journal of the American Association of Electrodiagnostic Medicine. 2004 Apr;29(4):465-83.26-34.
7. Karalliedde L, Baker D, Marrs TC. Organophosphate-induced intermediate syndrome: aetiology and relationships with myopathy. Toxicological reviews. 2006 Mar;25:1-4.
8. Umakanth M. Intermediate Syndrome Following Organophosphate Poisoning; Review Article. Asia Pacific Journal of Medical Toxicology. 2019 Jan 1;8(1).

9. Dekerlegand RL. Inspiratory muscle strength in adults with stable cystic fibrosis: Clinical predictors and the influence of disease severity. Rutgers The State University of New Jersey, School of Health Related Professions; 2014.

10. Mazzoli-Rocha F, Diniz CP, Rezende DP, Mendes FD, Hasslocher-Moreno AM, Sousa AS, Saraiva RM, Borghi-Silva A, Mediano MF, Alexandre DJ. Inspiratory Muscle Strength in Chagas Cardiomyopathy: A Systematic Scoping Review. Revista da Sociedade Brasileira de Medicina Tropical. 2023 Dec 8;56:e0389-2023.

11. Raghupathy V. *Role of Nitric Oxide and Mitochondria in Muscle Paralysis induced by Acute Organophosphate Poisoning* (Doctoral dissertation, The TamilnaduDr. MGR Medical University, Chennai).

12. Jaga K, Dharmani C. Sources of exposure to and public health implications of organophosphate pesticides. Revistapanamericana de saludpública. 2003;14:171-85.

13. Vogelmeier CF, Criner GJ, Martinez FJ, Anzueto A, Barnes PJ, Bourbeau J, Celli BR, Chen R, Decramer M, Fabbri LM, Frith P. Global strategy for the diagnosis, management, and prevention of chronic obstructive lung disease 2017 report. GOLD executive summary. American journal of respiratory and critical care medicine. 2017 Mar 1;195(5):557-82.

14. Karalliedde L, Baker D, Marrs TC. Organophosphate-induced intermediate syndrome: aetiology and relationships with myopathy. Toxicological reviews. 2006 Mar;25:1-4.

15. Yang CC, Deng JF. Intermediate syndrome following organophosphate insecticide poisoning. Journal of the Chinese Medical Association. 2007 Nov 1;70(11):467-72.

16. Alahakoon C, Dassanayake TL, Gawarammana IB, Sedgwick EM, Weerasinghe VS, Abdalla A, Roberts MS, Buckley NA. Prediction of organophosphorus insecticide-

induced intermediate syndrome with stimulated concentric needle single fibre electromyography. PloS one. 2018 Sep 27;13(9):e0203596.

17. Kumar R. *Organophosphate Poisoining Predicting the Need for Ventilatory Support* (Doctoral dissertation, Rajiv Gandhi University of Health Sciences (India)).
18. Akre C. The use of pyrethroids, carbamates, organophosphates, and other pesticides in veterinary medicine. Chemical Analysis of Non-antimicrobial Veterinary Drug Residues in Food. 2016 Nov 7:383-426.
19. Zeliger H. Human toxicology of chemical mixtures. William Andrew; 2011 Jun 24.
20. Nair PA, Sujatha CH. 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. 2012:259-309.
21. Umakanth M. Intermediate Syndrome Following Organophosphate Poisoning; Review Article. Asia Pacific Journal of Medical Toxicology. 2019 Jan 1;8(1).
22. Huang YT, Lai PC, Su CY, Chen YT, Cai CZ, Wang CH. Intermediate syndrome after organophosphate ingestion. Tzu Chi Medical Journal. 2007 Sep 1;19(3):159-63.
23. Alahakoon C, Dassanayake TL, Gawarammana IB, Weerasinghe VS. Can we predict intermediate syndrome? A review. Neurotoxicology. 2018 Dec 1;69:209-16.
24. Nirmalan N, Nirmalan M. Hormonal control of metabolism: regulation of plasma glucose. Anaesthesia & Intensive Care Medicine. 2020 Nov 1;21(11):578-83.
25. Haider R, Mehdi A, Zehra A, Das GK, Ahmed Z, Zameer S. Drug Development Research in Women. International Journal of Scientific Multidisciplinary Research. 2024 May 31;2(5):415-40.

26. Beckman D, Bonillas A, Diniz GB, Ott S, Roh JW, Elizaldi SR, Schmidt BA, Sammak RL, Van Rompay KK, Iyer SS, Morrison JH. SARS-CoV-2 infects neurons and induces neuroinflammation in a non-human primate model of COVID-19. Cell reports. 2022 Nov 1;41(5).

27. Ercin U. Oral Presentations Abstracts. Turk J Biochem. 2020;45(S2):25.

28. Baingane A. *A Novel Non-Enzymatic Glucose Biofuel Cell with Mobile Glucose Sensing Ankit Baingane* (Doctoral dissertation, Old Dominion University).

29. Adhami F. *Differential Adult and Neonatal Response to Cerebral Ischemia-Hypoxia* (Doctoral dissertation, University of Cincinnati).

30. Malik A, Dar TA, editors. The Glycome: Understanding the Diversity and Complexity of Glycobiology. CRC Press; 2021 Jul 7.

31. Dalby AJ, Bricknell OL, Opie LH. Effect of glucose-insulin-potassium infusions on epicardial ECG changes and on myocardial metabolic changes after coronary artery ligation in dogs. Cardiovascular Research. 1981 Oct 1;15(10):588-98.

32. Chen CJ, Buell TJ, Raper DM, Park MS, Kalani MY, Ironside N, James RF, Ding D. New biomarkers for the management of aneurysmal subarachnoid hemorrhage. Journal of Neurosurgery. 2019 Mar 22;130(5):1787-8.

33. Yin X, Ye G, Wang D. Middle cerebral artery tortuosity associated with aneurysm development. Journal of neurosurgery. 2018 Sep 7;130(5):1763-4.

34. Kumar A, Yadav YR. Factors predicting reoperation for chronic subdural hematoma. Journal of Neurosurgery. 2019 Mar 1;130(5):1784-6.

35. He HC, Dai Z. Prognostic molecular panel for skull base chordoma. Journal of Neurosurgery. 2019 Mar 15;130(5):1767-9. Tiwari S, Varma A, Vagha J, kanta Singh

U, Datta AK, Saboth P, Kumar V, Yadav B, Chattarjee S, Thakur A, Behera J. New Indian Journal of Pediatrics.

36. Tiwari S, Varma A, Vagha J, kanta Singh U, Datta AK, Saboth P, Kumar V, Yadav B, Chattarjee S, Thakur A, Behera J. New Indian Journal of Pediatrics.

37. Laghi F, Tobin MJ. Disorders of the respiratory muscles. American journal of respiratory and critical care medicine. 2003 Jul 1;168(1):10-48.

38. El-Nahhal Y, El-Nahhal I. Cardiotoxicity among Children Accidentally Exposed to Organophosphate Insecticides Coupled with Scorpion Bites in Gaza. Health. 2021 Sep 1;13(9):1045-63.

39. Mushtaq G, H Greig N, A Khan J, A Kamal M. Status of acetylcholinesterase and butyrylcholinesterase in Alzheimer's disease and type 2 diabetes mellitus. CNS & Neurological Disorders-Drug Targets (Formerly Current Drug Targets-CNS & Neurological Disorders). 2014 Oct 1;13(8):1432-9.

40. Demirtaş E, Korkmaz İ, Tekin YK, Demirtaş E, Çaltekin İ. Assessment of serum glucose/potassium ratio as a predictor for delayed neuropsychiatric syndrome of carbon monoxide poisoning. Human & Experimental Toxicology. 2021 Feb;40(2):207-13.

41. Wu XY, Zhuang YK, Cai Y, Dong XQ, Wang KY, Du Q, Yu WH. Serum glucose and potassium ratio as a predictive factor for prognosis of acute intracerebral hemorrhage. Journal of International Medical Research. 2021 Apr;49(4):03000605211009689.

42. Eddleston M, Szinicz L, Eyer P, Buckley N. Oximes in acute organophosphorus pesticide poisoning: a systematic review of clinical trials. Qjm. 2002 May 1;95(5):275-83.

43. Sharif AF, Fayed MM. Assessment of the serum glucose/potassium GLU/K ratio as a predictor of intermediate syndrome following acute anticholinesterase exposure. Neurotoxicology. 2022 Mar 1;89:161-73.

44. Lee DW, Gardner R, Porter DL, Louis CU, Ahmed N, Jensen M, Grupp SA, Mackall CL. Current concepts in the diagnosis and management of cytokine release syndrome. Blood, The Journal of the American Society of Hematology. 2014 Jul 10;124(2):188-95.

45. Sharif AF, Fayed MM. Assessment of the serum glucose/potassium GLU/K ratio as a predictor of intermediate syndrome following acute anticholinesterase exposure. Neurotoxicology. 2022 Mar 1;89:161-73.

46. Demirtaş E, Korkmaz İ, Tekin YK, Demirtaş E, Çaltekin İ. Assessment of serum glucose/potassium ratio as a predictor for delayed neuropsychiatric syndrome of carbon monoxide poisoning. Human & Experimental Toxicology. 2021 Feb;40(2):207-13.

47. Chen Y, Peng Y, Zhang X, Liao X, Lin J, Chen L, Lin Y. The blood glucose-potassium ratio at admission predicts in-hospital mortality in patients with acute type A aortic dissection. Scientific Reports. 2023 Sep 21;13(1):15707.

48. Boyuk F. The predictor potential role of the glucose to potassium ratio in the diagnostic differentiation of massive and non-massive pulmonary embolism. Clinical and Applied Thrombosis/Hemostasis. 2022 Feb;28:10760296221076146.

49. Lu Y, Ma X, Zhou X, Wang Y. The association between serum glucose to potassium ratio on admission and short-term mortality in ischemic stroke patients. Scientific Reports. 2022 May 17;12(1):8233.

50. El-Taftazani EA, Eweda SA. Serum glucose potassium ratio compared to theophylline level as a predictor of severity and outcome in acute theophylline toxicity. Zagazig Journal of Forensic Medicine and Toxicology. 2024 Jul 1;22(2):160-78.

51. Bouida W, Beltaief K, Msolli MA, Bzeouich N, Sekma A, Echeikh M, Mzali M, Boubaker H, Grissa MH, Boukef R, Hassine M. One-Year Outcome of Intensive Insulin Therapy Combined to Glucose-Insulin-Potassium in Acute Coronary Syndrome: A Randomized Controlled Study. Journal of the American Heart Association. 2017 Nov 14;6(11):e006674.

52. Marini JI, Sein ME. The role of the glucose potassium ratio in the management of traumatic brain injury. Korean Journal of Neurotrauma. 2023 Mar;19(1):82.

53. Yalçın G, Tunca H, Sayınbatur B, Anıl M. 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. Turkish archives of pediatrics. 2023 May;58(3):328.

54. Tongyoo S, Viarasilpa T, Permpikul C. Serum potassium levels and outcomes in critically ill patients in the medical intensive care unit. Journal of International Medical Research. 2018 Mar;46(3):1254-62.

55. Wahab NN, Cowden EA, Pearce NJ, Gardner MJ, Merry H, Cox JL, ICONS investigators. Is blood glucose an independent predictor of mortality in acute myocardial infarction in the thrombolytic era?. Journal of the American College of Cardiology. 2002 Nov 20;40(10):1748-54.

56. Zhang L, Li YH, Zhang HY, Chen ML, Gao MM, Hu AH, Yang HS, Yang HS. High-dose glucose-insulin-potassium treatment reduces myocardial apoptosis in

patients with acute myocardial infarction. European journal of clinical investigation. 2005 Mar;35(3):164-70.

57. Scott JF, Robinson GM, French JM, O'Connell JE, Alberti KG, Gray CS. Blood pressure response to glucose potassium insulin therapy in patients with acute stroke with mild to moderate hyperglycaemia. Journal of Neurology, Neurosurgery & Psychiatry. 2001 Mar 1;70(3):401-4.

58. Ntaios G, Egli M, Faouzi M, Michel P. J-shaped association between serum glucose and functional outcome in acute ischemic stroke. Stroke. 2010 Oct 1;41(10):2366-70.

59. Natarajan SK, Dandona P, Karmon Y, Yoo AJ, Kalia JS, Hao Q, Hsu DP, Hopkins LN, Fiorella DJ, Bendok BR, Nguyen TN. Prediction of adverse outcomes by blood glucose level after endovascular therapy for acute ischemic stroke. Journal of neurosurgery. 2011 Jun 1;114(6):1785-99.

60. Zhang J, Lv S, Jin T, Hu X. Logistic analysis of delayed reporting of emergency blood potassium and comparison of improved outcomes. Scientific Reports. 2024 Mar 13;14(1):6094.

61. Lin Z, Zhao Y, Xiao L, Qi C, Chen Q, Li Y. Blood urea nitrogen to serum albumin ratio as a new prognostic indicator in critical patients with chronic heart failure. ESC heart failure. 2022 Apr;9(2):1360-9.

62. Pan LN, Pan SA, Hong GL, Chen KW. A New Nomogram for Predicting 30-Day In-Hospital Mortality Rate of Acute Cholangitis Patients in the Intensive Care Unit. Emergency Medicine International. 2023;2023(1):9961438.

63. Kuo PJ, Huang CY, Hsu SY, Hsieh CH. Evaluating the prognostic value of the stress index in trauma patients. Heliyon. 2024 Sep 15;10(17).

64. Tsivgoulis, G., Katsanos, A.H., Mavridis, D., Lambadiari, V., Roffe, C., Macleod, M.J., Sevcik, P., Cappellari, M., Nevšímalová, M., Toni, D. and Ahmed, N., 2019. Association of baseline hyperglycemia with outcomes of patients with and without diabetes with acute ischemic stroke treated with intravenous thrombolysis: a propensity score–matched analysis from the SITS-ISTR registry. Diabetes, 68(9), pp.1861-1869.

65. Piironen K, Putaala J, Rosso C, Samson Y. Glucose and acute stroke: evidence for an interlude. Stroke. 2012 Mar;43(3):898-902.

66. Lashin HI, Sobeeh FG, Sobh ZK. Development and validation of a nomogram for predicting mechanical ventilation need among acutely intoxicated patients with impaired consciousness. Human & Experimental Toxicology. 2024 Jun 26;43:09603271241267214.

67. Huang CY, Rau CS, Huang CY, Su WT, Hsu SY, Hsieh CH. The Stress Index as a Predictor of Mortality in Patients with Isolated Moderate to Severe Traumatic Brain Injury. Diagnostics. 2024 Jun 13;14(12):1244.

68. Huang CY, Chou SE, Huang CY, Tsai CH, Hsu SY, Hsieh CH. Role of the stress index in predicting mortality among patients with traumatic femoral fractures. Diagnostics. 2024 Jul;14(14).

69. Li Y, Zhao L, Wang Y, Zhang X, Song J, Zhou Q, Sun Y, Yang C, Wang H. Development and validation of prediction models for neurocognitive disorders in adult patients admitted to the ICU with sleep disturbance. CNS Neuroscience & Therapeutics. 2022 Apr;28(4):554-65.

70. Patel VH, Vendittelli P, Garg R, Szpunar S, LaLonde T, Lee J, Rosman H, Mehta RH, Othman H. Neutrophil-lymphocyte ratio: a prognostic tool in patients with in-hospital cardiac arrest. World journal of critical care medicine. 2019 Feb 2;8(2):9.

71. Fishbein J, Coleman KM, Bhullar A, Sharma N, Zafeiropoulos S, Ansari U, Bimal T, Liu Y, Mountantonakis SE. Delayed cardiac repolarisation as a predictor of in-hospital mortality in patients with COVID-19. Heart. 2022 Oct 1;108(19):1539-46.

72. Vanhorebeek I, Langouche L, Van den Berghe G. Tight blood glucose control with insulin in the ICU: facts and controversies. Chest. 2007 Jul 1;132(1):268-78.

73. Poppe AY, Majumdar SR, Jeerakathil T, Ghali W, Buchan AM, Hill MD. Admission hyperglycemia predicts a worse outcome in stroke patients treated with intravenous thrombolysis. Diabetes care. 2009 Apr 1;32(4):617-22.

74. Rio A, Whelan K, Goff L, Reidlinger DP, Smeeton N. Occurrence of refeeding syndrome in adults started on artificial nutrition support: prospective cohort study. BMJ open. 2013 Jan 1;3(1):e002173.

75. Tan L, Xu Q, Shi R. A nomogram for predicting hospital mortality in intensive care unit patients with acute myocardial infarction. International Journal of General Medicine. 2021 Sep 18:5863-77.

76. Li N, Shao JM, Jiang Y, Wang CH, Li SB, Wang DC, Di WY. Unraveling the Predictors of Enlarged Perivascular Spaces: A Comprehensive Logistic Regression Approach in Cerebral Small Vessel Disease. International Journal of General Medicine. 2024 Dec 31:2513-25.

77. Sharif AF, Sobh ZK, Abdo SA, Alahmadi OM, Alharbi HA, Awaji MS, Alabdullatif FA, Baghlaf AM, Alanazi AF, Fayed MM. Evaluation of Global Dystonia Rating

Scale as a predictor of unfavorable outcomes among acute antipsychotics poisoned patients. Drug and chemical toxicology. 2024 Feb 9:1-8.

78. Yang F, Wang R, Lu W, Hu H, Li Z, Shui H. Prognostic value of blood urea nitrogen to serum albumin ratio for acute kidney injury and in-hospital mortality in intensive care unit patients with intracerebral haemorrhage: A retrospective cohort study using the MIMIC-IV database. BMJ open. 2023 Aug 1;13(8):e069503.

79. Demchuk AM, Tanne D, Hill MD, Kasner SE, Hanson S, Grond M, Levine SR, Multicentre tPA Stroke Survey Group. Predictors of good outcome after intravenous tPA for acute ischemic stroke. Neurology. 2001 Aug 14;57(3):474-80.

80. Park J, Kim M, Kim JW, Choi HJ, Hong SH. Predictive Value of the D-Dimer-to-Fibrinogen Ratio for Acute Kidney Injury after Living-Donor Liver Transplantation: A Retrospective Observational Cohort Study Using Logistic Regression and Propensity Score Matching Analyses. Journal of Clinical Medicine. 2024 Sep 17;13(18):5499.

81. Van der Horst IC, Nijsten MW, Vogelzang M, Zijlstra F. Persistent hyperglycemia is an independent predictor of outcome in acute myocardial infarction. Heart Views. 2007 Jun 1;8(2):43-51.

82. Yang CJ, Liao WI, Wang JC, Tsai CL, Lee JT, Peng GS, Lee CH, Hsu CW, Tsai SH. Usefulness of glycated hemoglobin A1c-based adjusted glycemic variables in diabetic patients presenting with acute ischemic stroke. The American journal of emergency medicine. 2017 Sep 1;35(9):1240-6.

83. Khitan Z, Shapiro AP, Shah PT, Sanabria JR, Santhanam P, Sodhi K, Abraham NG, Shapiro JI. Predicting adverse outcomes in chronic kidney disease using machine learning methods: data from the modification of diet in renal disease. Marshall Journal of Medicine. 2017;3(4):68.

84. KARADUMAN A, YILMAZ C, KETEN MF, BALABAN I, GUNER ZE, CELIK M. The Relationship Between the Modified Glasgow Prognostic and SYNTAX Scores in Patients with Non-ST Elevation Myocardial Infarction. Inflammation. 2024;22:4.

85. Bruno A, Levine SR, Frankel MR, Brott TG, Lin Y, Tilley BC, Lyden PD, Broderick JP, Kwiatkowski TG, Fineberg SE, NINDS rt-PA Stroke Study Group*. Admission glucose level and clinical outcomes in the NINDS rt-PA Stroke Trial. Neurology. 2002 Sep 10;59(5):669-74.

86. Yuval Bar-Asher S, Shefer A, Shoham-Vardi I, Sergienko R, Wolak A, Sheiner E, Wolak T. Routine blood tests during pregnancy for predicting future increases in risk of cardiovascular morbidity. International Journal of Gynecology& Obstetrics. 2018 Nov;143(2):178-83.

87. Zhao N, Hu W, Wu Z, Wu X, Li W, Wang Y, Zhao H. The red blood cell distribution width–albumin ratio: a promising predictor of mortality in stroke patients. International journal of general medicine. 2021 Jul 22:3737-47.

88. Njim T, Tanyitiku BS. Prognostic models for the clinical management of malaria and its complications: a systematic review. BMJ open. 2019 Nov 1;9(11):e030793.

89. Li N, Hu YD, Jiang Y, Ling L, Wang CH, Shao JM, Li SB, Di WY. Integrating clinical and biochemical markers: a novel nomogram for predicting lacunes in cerebral small vessel disease. Frontiers in Aging Neuroscience. 2024 Aug 23;16:1404836.

90. Kruyt ND, Roos YW, Mees SD, Van den Bergh WM, Algra A, Rinkel GJ, Biessels GJ. High mean fasting glucose levels independently predict poor outcome and delayed cerebral ischaemia after aneurysmal subarachnoid haemorrhage. Journal of Neurology, Neurosurgery & Psychiatry. 2008 Dec 1;79(12):1382-5.

91. Li F, Xin H, Zhang J, Fu M, Zhou J, Lian Z. Prediction model of in-hospital mortality in intensive care unit patients with heart failure: machine learning-based, retrospective analysis of the MIMIC-III database. BMJ open. 2021 Jul 1;11(7):e044779.

92. Yong M, Kaste M. Dynamic of hyperglycemia as a predictor of stroke outcome in the ECASS-II trial. Stroke. 2008 Oct 1;39(10):2749-55.

93. Lipshutz AK, Gropper MA, Warner DS, Warner MA. Perioperative glycemic control: an evidence-based review. The Journal of the American Society of Anesthesiologists. 2009 Feb 1;110(2):408-21.

94. Bae, D.H., Lee, H.Y., Jung, Y.H., Jeung, K.W., Lee, B.K., Youn, C.S., Kang, B.S., Heo, T. and Min, Y.I., 2021. PROLOGUE (PROgnostication using LOGistic regression model for Unselected adult cardiac arrest patients in the Early stages): Development and validation of a scoring system for early prognostication in unselected adult cardiac arrest patients. Resuscitation, 159, pp.60-68.

95. Chamorro Á, Brown S, Amaro S, Hill MD, Muir KW, Dippel DW, Van Zwam W, Butcher K, Ford GA, Den Hertog HM, Mitchell PJ. Glucose modifies the effect of endovascular thrombectomy in patients with acute stroke: A pooled-data meta-analysis. Stroke. 2019 Mar;50(3):690-6.

96. Zhu P, Lu L, Xu Y, Greyson C, Schwartz GG. Glucose-insulin-potassium preserves systolic and diastolic function in ischemia and reperfusion in pigs. American Journal of Physiology-Heart and Circulatory Physiology. 2000 Feb 1;278(2):H595-603.

97. Lewis KS, Kane-Gill SL, Bobek MB, Dasta JF. Intensive insulin therapy for critically III patients. Annals of Pharmacotherapy. 2004 Jul;38(7-8):1243-51.

98. Tabak YP, Sun X, Nunez CM, Gupta V, Johannes RS. Predicting readmission at early hospitalization using electronic clinical data: an early readmission risk score. Medical care. 2017 Mar 1;55(3):267-75.

99. Sidhu GK, Singh S, Kumar V, Dhanjal DS, Datta S, Singh J. Toxicity, monitoring and biodegradation of organophosphate pesticides: a review. Critical reviews in environmental science and technology. 2019 Jul 3;49(13):1135-87.

100. Iwuozor KO, Emenike EC, Gbadamosi FA, Ighalo JO, Umenweke GC, Iwuchukwu FU, Nwakire CO, Igwegbe CA. Adsorption of organophosphate pesticides from aqueous solution: a review of recent advances. International Journal of Environmental Science and Technology. 2023 May;20(5):5845-94.

101. Suratman S, Edwards JW, Babina K. Organophosphate pesticides exposure among farmworkers: pathways and risk of adverse health effects. Reviews on environmental health. 2015 Mar 1;30(1):65-79.

102. Soltaninejad K, Shadnia S. History of the use and epidemiology of organophosphorus poisoning. Basic and clinical toxicology of organophosphorus compounds. 2014:25-43.

103. Muñoz-Quezada MT, Lucero BA, Iglesias VP, Muñoz MP, Cornejo CA, Achu E, Baumert B, Hanchey A, Concha C, Brito AM, Villalobos M. Chronic exposure to organophosphate (OP) pesticides and neuropsychological functioning in farm workers: a review. International journal of occupational and environmental health. 2016 Jan 2;22(1):68-79.

104. Mdeni NL, Adeniji AO, Okoh AI, Okoh OO. Analytical evaluation of carbamate and organophosphate pesticides in human and environmental matrices: a review. Molecules. 2022 Jan 18;27(3):618.

105. Ranjan A, Jindal T. Toxicology of organophosphate poisoning. Springer International Publishing; 2022.

106. Kaushal J, Khatri M, Arya SK. A treatise on Organophosphate pesticide pollution: Current strategies and advancements in their environmental degradation and elimination. Ecotoxicology and Environmental Safety. 2021 Jan 1;207:111483.

107. Alozi M, Rawas-Qalaji M. Treating organophosphates poisoning: management challenges and potential solutions. Critical reviews in toxicology. 2020 Oct 20;50(9):764-79.

108. Kavvalakis MP, Tsatsakis AM. The atlas of dialkylphosphates; assessment of cumulative human organophosphorus pesticides' exposure. Forensic Science International. 2012 May 10;218(1-3):111-22.

109. Muñoz-Quezada MT, Lucero B, Iglesias V, Levy K, Muñoz MP, Achú E, Cornejo C, Concha C, Brito AM, Villalobos M. Exposure to organophosphate (OP) pesticides and health conditions in agricultural and non-agricultural workers from Maule, Chile. International journal of environmental health research. 2017 Jan 2;27(1):82-93.

110. Perry J, Cotton J, Rahman MA, Brumby SA. Organophosphate exposure and the chronic effects on farmers: a narrative review. Rural and Remote Health. 2020 Mar;20(1):206-22.

111. Yadav IC, Devi NL, Syed JH, Cheng Z, Li J, Zhang G, Jones KC. Current status of persistent organic pesticides residues in air, water, and soil, and their possible effect on neighboring countries: A comprehensive review of India. Science of the Total Environment. 2015 Apr 1;511:123-37.

112. Sharif AF, Fayed MM. Assessment of the serum glucose/potassium GLU/K ratio as a predictor of intermediate syndrome following acute anticholinesterase exposure. Neurotoxicology. 2022 Mar 1;89:161-73.

113. Elmansy AM, Hannora DM, Khalifa HK. Serum glucose/potassium ratio as an indicator of early and delayed outcomes of acute carbon monoxide poisoning. Toxicology Research. 2024 Oct;13(5):tfae168.

114. El-Sarnagawy GN, Hafez AS, Ghonem MM. Role of serum glucose/potassium ratio in assessing poisoning severity and adverse outcomes in patients with acute aluminum phosphide poisoning. Toxicology Reports. 2025 Feb 4:101947.

115. Yalçın G, Tunca H, Sayınbatur B, Anıl M. 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. Turkish archives of pediatrics. 2023 May 1;58(3):328.

116. Liu J, Luo F, Guo Y, Li Y, Jiang C, Pi Z, Luo J, Long Z, Wen J, Huang Z, Zhu J. Association between serum glucose potassium ratio and mortality in critically ill patients with intracerebral hemorrhage. Scientific Reports. 2024 Nov 9;14(1):27391.

117. Boyuk F. The predictor potential role of the glucose to potassium ratio in the diagnostic differentiation of massive and non-massive pulmonary embolism. Clinical and Applied Thrombosis/Hemostasis. 2022 Feb;28:10760296221076146.

118. Elmansy AM, Hannora DM, Khalifa HK. Serum glucose/potassium ratio as an indicator of early and delayed outcomes of acute carbon monoxide poisoning. Toxicology Research. 2024 Oct;13(5):tfae168.

119. Chen Y, Peng Y, Zhang X, Liao X, Lin J, Chen L, Lin Y. The blood glucose-potassium ratio at admission predicts in-hospital mortality in patients with acute type A aortic dissection. Scientific Reports. 2023 Sep 21;13(1):15707.

120. El-Taftazani EA, Eweda SA. Serum glucose potassium ratio compared to theophylline level as a predictor of severity and outcome in acute theophylline toxicity. Zagazig Journal of Forensic Medicine and Toxicology. 2024 Jul 1;22(2):160-78.

121. Marini JI, Sein ME. The role of the glucose potassium ratio in the management of traumatic brain injury. Korean Journal of Neurotrauma. 2023 Mar 15;19(1):82.'

122. Scott JF, Robinson GM, French JM, O'Connell JE, Alberti KG, Gray CS. Blood pressure response to glucose potassium insulin therapy in patients with acute stroke with mild to moderate hyperglycaemia. Journal of Neurology, Neurosurgery & Psychiatry. 2001 Mar 1;70(3):401-4.

123. Lashin HI, Sobeeh FG, Sobh ZK. Development and validation of a nomogram for predicting mechanical ventilation need among acutely intoxicated patients with impaired consciousness. Human & Experimental Toxicology. 2024 Jun 26;43:09603271241267214.

124. Kuo PJ, Huang CY, Hsu SY, Hsieh CH. Evaluating the prognostic value of the stress index in trauma patients. Heliyon. 2024 Sep 15;10(17).

125. Huang CY, Chou SE, Huang CY, Tsai CH, Hsu SY, Hsieh CH. Role of the stress index in predicting mortality among patients with traumatic femoral fractures. Diagnostics. 2024 Jul 12;14(14):1508.

126. Ramadori GP. Organophosphorus poisoning: Acute respiratory distress syndrome (ARDS) and cardiac failure as cause of death in hospitalized patients. International journal of molecular sciences. 2023 Apr 3;24(7):6658.

127. Klainbart S, Kelmer E, Chai O, Segev G, Aroch I. Prevalence, clinical manifestations, laboratory findings, treatment, and outcome of intermediate syndrome in anticholinesterase pesticide intoxication of dogs: A retrospective study. The Veterinary Journal. 2022 Sep 1;287:105883.

128. Mohammed E, Ali A, Abd El Wahab M, Mahmoud M. The Potential Role Of Glial Fibrillary Acidic Protein In Evaluation of Organophosphorus-Induced Neurotoxicity: A prospective clinical study. Ain Shams Journal of Forensic Medicine and Clinical Toxicology. 2023 Jan 1;40(1):101-11.

ANNEXURE – I

ETHICAL CLEARANCE CERTIFICATE

Azadi Ka Amrit Mahotsav

BLDE
(DEEMED TO BE UNIVERSITY)
Declared as Deemed to be University u/s 3 of UGC Act, 1956
Accredited with 'A' Grade by NAAC (Cycle-2)
The Constituent College
SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPURA

BLDE (DU)/IEC/ 903/2023-24 10/4/2023

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this University met on **Saturday, 18th March, 2023 at 11.30 a.m. in the CAL Laboratory, Dept. of Pharmacology,** scrutinizes the Synopsis/ Research Projects of Post Graduate Student / Under Graduate Student /Faculty members of this University /Ph.D. Student College from ethical clearance point of view. After scrutiny, the following original/ corrected and revised version synopsis of the thesis/ research projects has been accorded ethical clearance.

TITLE: "EFFECTIVENESS OF SERUM GLUCOSE/POTASSIUM RATIO AS A TOOL FOR PREDICTOR OF INTERMEDIATE SYNDROME".

NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR: DR.VISHNU J NAIR

NAME OF THE GUIDE: DR.RAVI B. PATIL, PROFESSOR AND HOD, DEPT. OF EMERGENCY MEDICINE AND CRITICAL CARE.

Dr. Santoshkumar Jeevangi
Chairperson
IEC, BLDE (DU),
VIJAYAPURA

Chairman,
Institutional Ethical Committee,
BLDE (Deemed to be University)
Vijayapura

Dr.Akram A. Naikwadi
Member Secretary
IEC, BLDE (DU),
VIJAYAPURA

MEMBER SECRETARY
Institutional Ethics Committee
BLDE (Deemed to be University)
Vijayapura-586103, Karnataka

Following documents were placed before Ethical Committee for Scrutinization.

- Copy of Synopsis/Research Projects
- Copy of inform consent form
- Any other relevant document

Smt. Bangaramma Sajjan Campus, B. M. Patil Road (Sholapur Road), Vijayapura - 586103, Karnataka, India.
BLDE (DU): Phone: +918352-262770, Fax: +918352-263303, Website: www.bldedu.ac.in, E-mail:office@bldedu.ac.in
College: Phone: +918352-262770, Fax: +918352-263019, E-mail: bmpmc.principal@bldedu.ac.in

ANNEXURE II

INFORMED CONSENT FORM

BLDEDU'S SHRI B.M. PATIL MEDICAL COLLEGE
HOSPITAL AND RESEARCH CENTRE,

VIJAYAPURA-586103

TITLE OF THE PROJECT –

"SERUM GLUCOSE/POTTASSIUM RATIO AS A TOOL FOR PROGNOSIS OF ORGANOPHOSPHATE POISONING AND PREDICTOR OF INTERMEDIATE SYNDROME."

PRINCIPAL INVESTIGATOR -DR. VISHNU J NAIR

P.G. GUIDE NAME - DR. BABU P KATTIMANI

CHAIRMANETHICALCOMMITTEE

All aspects of this consent form are explained to the patient in the language understood by him/her.

1) PURPOSEOFRESEARCH:

I have been informed about this study. I have also been given a free choice of participation in

this study.

2) PROCEDURE:

I am aware that in addition to the routine care received, I will be asked a series of questions by the investigator. I have been asked to undergo the necessary investigations and treatment, which will help the investigator in this study.

2) Risk and Discomforts

I understand that I may experience some pain and discomfort during the examination or treatment. This is mainly the result of my condition, and the procedure of this study is not expected to exaggerate these feelings that are associated with the usual course of treatment.

3) Benefits

I understand that participation in this study will help improve patients' survival and overall outcomes.

4) Confidentiality

I understand that the medical information produced by this study will become part of the hospital records and will be subject to confidentiality and privacy regulations. Information of a sensitive personal nature will not be included in the medical records but will be stored in the investigator's research file, identified only by a code number. The code-key linking my name to the study will be kept in a separate location. If data from this study is used for publication or teaching purposes, my name will not be used. Any photographs, audio, or video recordings will only be used with my special written permission. I will have the opportunity to review these materials before giving consent for their use.

5) Request for More Information

I understand that I may ask questions about the study at any time. Dr. Shubham B. Deore is available to answer my questions or concerns. I will also be informed of any significant new findings discovered during the study that might influence my continued participation. If I wish to discuss my participation or concerns with someone not directly involved in the study, I am aware that the hospital's social worker is available for consultation. A copy of this consent form will be given to me for careful reading and reference.

6) Refusal or Withdrawal of Participation

I understand that participation in this study is voluntary. I may refuse to participate or withdraw my consent at any time without affecting my present or future care at this hospital. I also understand that Dr. Shubham may terminate my participation in the study after

explaining the reasons and will assist in arranging continued care through my physician or physical therapist, if necessary.

7) Injury Statement

I understand that in the unlikely event of injury resulting directly from my participation in this study, appropriate treatment will be provided if the injury is reported promptly. However, no further compensation will be provided. I acknowledge that by agreeing to participate in this study, I am not waiving any of my legal rights. The purpose of the research, required procedures, possible risks, and benefits have been explained to me in detail and in a language I understand.

DR.VISHNU J NAIR
(Investigator)
Date

II) STUDY SUBJECT CONSENT STATEMENT

I confirm that **Dr. Vishnu J. Nair** has explained to me the purpose of the research, the study procedures that I will undergo, and the possible risks, discomforts, and benefits that I may experience in my own language. I have read the form and understand this consent.
Therefore, I agree to give my consent to participate as a subject in this research project.

_____

_____

Participant/ Guardian Date:

_____

Witness to signature Date:

ANNEXURE III

B.L.D.E (DEEMED TO BE UNIVERSITY)

SHRI B M PATIL MEDICAL COLLEGE,

VIJAYAPURA, KARNATAKA

SCHEME OF CASE TAKING

INFORMANT:

Name: CASENO:

Age: IP NO:

Sex: DOA:

Religion:

Past Occupation:

Present Occupation:

Contact Number:

Residence:

ESI criteria-Diagnosis –

Hospital admission –YES/NO

ICU admission –YES: Need for non-invasive ventilation /Need for invasive ventilation/NO: Length of hospital stay–

Emergencyward____/ICCU_____

Causeofdeath(ifdied) –

48-hrsoutcome:Improved/Deteriorated/Died

	At time of admission	At12thhrsof admission	At 24 hour Of admission	Atthe48thhr saftera dmissi on
Serum Glucose				
Shock Potassium				

CURRICULUM-VITAE

NAME DR.BABU KATTIMANI

DESIGNATION PROFESSOR

DEPARTMENT OF EMERGENCY MEDICINE

CONTACT: 9342594480

EDUCATION M.S.GENERAL SURGERY,

PRESENT DESIGNATION: PROFESSOR,

DEPARTMENT OF EMERGENCY MEDICINE

SHRI B M PATIL MEDICAL COLLEGE AND

RESEARCH CENTER

VIJAYAPURA, KARNATAKA

BIO-DATA

INVESTIGATOR NAME: DR VISHNU J NAIR

QUALIFICATION: M.B.B.S

KARNATAKA MEDICAL

COUNCIL NUMBER: 158004

ADDRESS: Thenganal House, Kondadu ,Ramapuram Bazar P O ,Kottayam District, Kerala,

India

PHONE NUMBER: 7907041599

ANNEXURE III

MASTER CHART

Age	Sex	IMS +/-	Occupation	Residence	Exposure	Causes
36	m	IMS-	farmer	Urban	Monochrotophous	Suicide
39	m	IMS-	farmer	Urban	Profenofos	Suicide
37	m	IMS-	farmer	Urban	Profenofos	Suicide
41	m	IMS+	farmer	Urban	Malathion	Suicide
38	m	IMS-	farmer	Urban	Profenofos	Suicide
42	m	IMS-	Civil E	Urban	Monochrotophous	Suicide
37	m	IMS-	Civil E	Urban	Profenofos	Suicide
45	f	IMS-	HW	Urban	Monochrotophous	Suicide
38	f	IMS-	HW	Rural	Chlorpyrifos	Suicide
39	f	IMS-	HW	Rural	Monochrotophous	Accidental
44	f	IMS-	HW	Rural	Unknown	Accidental
38	f	IMS-	HW	Rural	Profenofos	Accidental
42	f	IMS+	HW	Rural	Dimethoate	Accidental
23	f	IMS-	HW	Rural	Chlorpyrifos	Accidental
21	m	IMS-	student	Rural	Monochrotophous	Suicide
18	m	IMS-	student	Rural	Unknown	Suicide
19	m	IMS-	student	Rural	Monochrotophous	Suicide
21	m	IMS-	student	Rural	Malathion	Suicide
19	m	IMS-	student	Rural	Monochrotophous	Suicide
23	m	IMS-	student	Rural	Profenofos	Suicide
21	m	IMS-	student	Rural	Monochrotophous	Suicide
18	m	IMS-	unemployed	Rural	Chlorpyrifos	Suicide
19	m	IMS-	unemployed	Rural	Profenofos	Suicide
21	f	IMS-	HW	Rural	Monochrotophous	Suicide
19	f	IMS-	HW	Rural	Unknown	Suicide
19	f	IMS-	HW	Rural	Monochrotophous	Suicide
21	f	IMS+	HW	Rural	Chlorpyrifos	Suicide
19	f	IMS-	HW	Rural	Profenofos	Suicide
21	f	IMS+	HW	Rural	Dimethoate	Suicide
18	f	IMS-	HW	Rural	Monochrotophous	Suicide
19	f	IMS-	HW	Rural	Monochrotophous	Suicide
21	m	IMS-	farmer	Rural	Monochrotophous	Suicide
30	m	IMS-	farmer	Rural	Monochrotophous	Suicide
34	m	IMS-	farmer	Rural	Unknown	Suicide
32	m	IMS+	farmer	Rural	Dimethoate	Suicide
25	m	IMS-	farmer	Rural	Profenofos	Suicide

28		m		IMS-		farmer				Rural			Monochrotophous	Suicide
29		m		IMS-		farmer				Rural			Malathion	Suicide
30		m		IMS+		farmer				Rural			Monochrotophous	Suicide
26		m		IMS-		manual				Rural			Unknown	Suicide
28		m		IMS+		manual				Rural			Dimethoate	Suicide
34		f		IMS-		HW				Rural			Profenofos	Suicide
27		f		IMS-		HW				Rural			Monochrotophous	Suicide
30		m		IMS-		manual				Rural			Malathion	Suicide
34		f		IMS-		HW				Rural			Monochrotophous	Suicide
32		f		IMS-		HW				Rural			Unknown	Suicide
25		f		IMS-		HW				Rural			Profenofos	Suicide
28		f		IMS-		HW				Rural			Monochrotophous	Suicide
29		m		IMS-		manual				Rural			Dimethoate	Accidental
30		m		IMS-		manual				Rural			Monochrotophous	Accidental
26		f		IMS-		HW				Rural			Monochrotophous	Accidental
28		f		IMS-		HW				Rural			Chlorpyrifos	Accidental
34		f		IMS-		HW				Rural			Profenofos	Suicide
27		f		IMS-		HW				Rural			Monochrotophous	Suicide
28		f		IMS-		HW				Rural			Monochrotophous	Suicide
29		m		IMS-		manual				Rural			Malathion	Suicide
30		m		IMS-		manual				Rural			Profenofos	Suicide
26		m		IMS-		manual				Rural			Monochrotophous	Suicide
34		m		IMS+		manual				Rural			Profenofos	Suicide
29		f		IMS-		HW				Rural			Chlorpyrifos	Accidental
47		f		IMS+		HW				Rural			Monochrotophous	Accidental
49		f		IMS-		HW				Rural			Monochrotophous	Accidental
51		f		IMS-		HW				Rural			Malathion	Accidental
53		f		IMS+		HW				Rural			Dimethoate	Accidental
59		m		IMS-		manual				Rural			Monochrotophous	Accidental
48		m		IMS-		manual				Rural			Chlorpyrifos	Accidental
54		m		IMS-		manual				Rural			Monochrotophous	Accidental
49		m		IMS+		manual				Rural			Monochrotophous	Accidental
52		f		IMS-		HW				Rural			Unknown	Accidental
51		f		IMS-		HW				Rural			Monochrotophous	Accidental
47		f		IMS-		HW				Rural			Dimethoate	Accidental
49		f		IMS+		HW				Rural			Chlorpyrifos	Accidental
51		f		IMS-		HW				Rural			Malathion	Suicide
53		f		IMS-		HW				Rural			Profenofos	Suicide
59		f		IMS-		HW				Urban			Monochrotophous	Suicide
48		f		IMS+		HW				Urban			Malathion	Suicide
21		f		IMS-		HW				Urban			Unknown	Occupational
19		f		IMS-		HW				Urban			Chlorpyrifos	Occupational
19		f		IMS+		HW				Urban			Dimethoate	Occupational
18		m		IMS-		unemployed				Urban			Profenofos	Occupational

19		m		IMS-		unemployed			Urban			Monochrotophous	Suicide
21		m		IMS-		unemployed			Urban			Malathion	Suicide
59		m		IMS-		unemployed			Urban			Monochrotophous	Suicide
71		m		IMS-		unemployed			Urban			Unknown	Suicide
66		f		IMS-		unemployed			Urban			Dimethoate	Suicide
62		f		IMS-		unemployed			Urban			Profenofos	Suicide
63		m		IMS-		unemployed			Urban			Chlorpyrifos	Suicide
75		m		IMS-		unemployed			Urban			Monochrotophous	Suicide
66		m		IMS-		unemployed			Urban			Dimethoate	Suicide
62		m		IMS-		unemployed			Urban			Monochrotophous	Suicide
63		m		IMS+		unemployed			Urban			Monochrotophous	Suicide
56		m		IMS+		unemployed			Urban			Malathion	Suicide
59		m		IMS-		manual			Urban			Profenofos	Suicide
71		m		IMS+		manual			Urban			Malathion	Suicide
66		m		IMS-		manual			Urban			Chlorpyrifos	Suicide
62		m		IMS-		manual			Urban			Monochrotophous	Suicide
63		m		IMS-		manual			Urban			Unknown	Suicide
75		m		IMS-		manual			Urban			Chlorpyrifos	Occupational
66		m		IMS+		Civil E			Urban			Dimethoate	Occupational
21		m		IMS-		Civil E			Urban			Profenofos	Suicide
19		m		IMS-		Civil E			Urban			Monochrotophous	Suicide
18		m		IMS-		manual			Urban			Malathion	Suicide
19		m		IMS+		manual			Urban			Malathion	Suicide
21		f		IMS-		HW			Rural			Monochrotophous	Suicide
21		f		IMS-		HW			Rural			Profenofos	Occupational
19		f		IMS-		HW			Rural			Monochrotophous	Occupational
19		f		IMS-		HW			Rural			Chlorpyrifos	Suicide
21		f		IMS-		HW			Rural			Monochrotophous	Suicide
19		f		IMS-		Civil E			Rural			Profenofos	Suicide
21		f		IMS+		Civil E			Rural			Malathion	Suicide
18		f		IMS-		Civil E			Rural			Unknown	Occupational
19		f		IMS+		Civil E			Rural			Malathion	Occupational
21		f		IMS-		Civil E			Rural			Profenofos	Occupational
19		f		IMS-		Civil E			Rural			Monochrotophous	Occupational
23		f		IMS-		Civil E			Rural			Unknown	Occupational
21		f		IMS-		Civil E			Rural			Monochrotophous	Occupational
18		f		IMS-		Civil E			Rural			Profenofos	Occupational
19		f		IMS-		Civil E			Rural			Monochrotophous	Occupational
21		m		IMS-		farmer			Rural			Malathion	
19		m		IMS-		farmer			Rural			Monochrotophous	Suicide
19		m		IMS-		farmer			Rural			Malathion	Suicide
18		m		IMS-		farmer			Rural			Profenofos	Suicide
19		m		IMS-		farmer			Rural			Malathion	Suicide
21		m		IMS-		farmer			Rural			Profenofos	Suicide

19		m		IMS-		farmer				Rural			Chlorpyrifos	Suicide
19		m		IMS-		unemployed				Rural			Malathion	Suicide
21		m		IMS-		unemployed				Rural			Monochrotophous	Occupational
19		m		IMS-		unemployed				Urban			Unknown	Occupational
19		m		IMS-		unemployed				Urban			Profenofos	Accidental
18		m		IMS-		unemployed				Urban			Monochrotophous	Occupational
19		m		IMS-		unemployed				Urban			Malathion	Occupational
21		m		IMS-		farmer				Urban			Dimethoate	Suicide
21		m		IMS-		farmer				Urban			Monochrotophous	Suicide
19		m		IMS-		farmer				Urban			Unknown	Suicide
19		m		IMS-		farmer				Urban			Profenofos	Occupational
18		m		IMS-		farmer				Urban			Monochrotophous	Occupational
19		m		IMS-		farmer				Urban			Malathion	
21		m		IMS-		farmer				Urban			Chlorpyrifos	Suicide
19		f		IMS-		farmer				Urban			Profenofos	Suicide
18		f		IMS-		farmer				Urban			Monochrotophous	Suicide
21		f		IMS-		manual				Urban			Unknown	Suicide
19		f		IMS-		manual				Urban			Dimethoate	Suicide
18		f		IMS-		manual				Urban			Profenofos	Suicide
19		f		IMS-		manual				Urban			Monochrotophous	Accidental
21		f		IMS-		manual				Urban			Malathion	Accidental
21		f		IMS-		manual				Rural			Dimethoate	Accidental
19		f		IMS-		manual				Rural			Profenofos	Accidental
21		f		IMS-		manual				Rural			Monochrotophous	Accidental
21		f		IMS-		manual				Rural			Chlorpyrifos	Accidental
23		f		IMS-		manual				Rural			Monochrotophous	Accidental
21		f		IMS-		manual				Rural			Malathion	Accidental
18		f		IMS-		unemployed				Rural			Profenofos	Accidental
19		f		IMS-		unemployed				Rural			Monochrotophous	Accidental
21		m		IMS-		unemployed				Rural			Malathion	Accidental
19		m		IMS-		manual				Rural			Profenofos	Accidental
23		m		IMS-		manual				Rural			Monochrotophous	Accidental
21		m		IMS-		manual				Rural			Malathion	Accidental
18		m		IMS-		manual				Rural			Malathion	Accidental
19		m		IMS-		manual				Rural			Profenofos	Accidental
21		m		IMS-		manual				Rural			Monochrotophous	Accidental
19		m		IMS-		manual				Rural			Malathion	Accidental
19		m		IMS-		Civil E				Rural			Chlorpyrifos	Accidental
26		m		IMS-		Civil E				Rural			Profenofos	Accidental
28		m		IMS-		Civil E				Rural			Unknown	Accidental
34		m		IMS-		Civil E				Rural			Monochrotophous	Accidental
27		m		IMS-		Civil E				Rural			Malathion	Accidental
30		m		IMS-		Civil E				Rural			Profenofos	Occupational
34		m		IMS-		Civil E				Rural			Monochrotophous	Occupational

32		m		IMS-		Civil E				Rural			Chlorpyrifos	Suicide
25		f		IMS-		farmer				Rural			Profenofos	Suicide
28		f		IMS-		farmer				Rural			Malathion	Suicide
29		f		IMS-		farmer				Rural			Chlorpyrifos	Suicide
30		f		IMS-		farmer				Rural			Profenofos	Suicide
34		f		IMS-		manual				Rural			Malathion	Suicide
27		m		IMS-		manual				Rural			Chlorpyrifos	Suicide
30		m		IMS-		manual				Rural			Profenofos	Suicide
34		m		IMS-		manual				Rural			Malathion	Suicide
25		m		IMS-		manual				Rural			Malathion	Suicide
28		f		IMS-		Civil E				Rural			Profenofos	Suicide
29		f		IMS-		Civil E				Rural			Chlorpyrifos	Accidental
30		f		IMS-		Civil E				Rural			Profenofos	Accidental
34		f		IMS-		farmer				Rural			Malathion	Accidental
30		f		IMS-		farmer				Rural			Profenofos	Accidental
34		f		IMS-		unemployed				Rural			Unknown	Accidental
26		f		IMS-		manual				Rural			Chlorpyrifos	Accidental
28		f		IMS-		manual				Rural			Profenofos	Accidental
34		m		IMS-		manual				Rural			Malathion	Accidental
27		m		IMS-		manual				Rural			Chlorpyrifos	Accidental
19		m		IMS-		manual				Rural			Unknown	Accidental
23		m		IMS-		manual				Rural			Chlorpyrifos	Accidental
21		m		IMS-		manual				Rural			Profenofos	Accidental
18		m		IMS-		manual				Rural			Malathion	Accidental
19		m		IMS-		manual				Rural			Chlorpyrifos	Accidental
21		f		IMS-		manual				Rural			Unknown	Accidental
19		f		IMS-		manual				Rural			Malathion	Accidental
19		m		IMS-		manual				Rural			Dimethoate	Accidental
18		m		IMS-		manual				Rural			Chlorpyrifos	Occupational
19		m		IMS-		manual				Rural			Malathion	Occupational
21		m		IMS-		manual				Rural			Profenofos	Occupational
19		m		IMS-		manual				Rural			Chlorpyrifos	Occupational
19		m		IMS-		manual				Rural			Malathion	Occupational
21		m		IMS-		manual				Rural			Chlorpyrifos	Occupational
19		m		IMS-		manual				Rural			Malathion	Occupational
19		m		IMS-		manual				Rural			Profenofos	Occupational
18		m		IMS-		manual				Rural			Chlorpyrifos	Occupational
19		m		IMS-		manual				Rural			Malathion	Occupational
21		m		IMS-		manual				Rural			Dimethoate	Occupational
21		m		IMS-		manual				Rural			Chlorpyrifos	Occupational
19		m		IMS-		manual				Rural			Profenofos	Occupational
19		f		IMS-		manual				Rural			Chlorpyrifos	Occupational
18		f		IMS-		manual				Urban			Chlorpyrifos	Occupational
19		f		IMS-		manual				Urban			Malathion	Occupational

37		f		IMS-		manual				Urban			Chlorpyrifos	Occupational
45		f		IMS-		manual				Urban			Unknown	Occupational
38		f		IMS-		manual				Urban			Profenofos	Occupational
39		m		IMS-		manual				Urban			Chlorpyrifos	Occupational
44		m		IMS-		manual				Urban			Malathion	Occupational
36		m		IMS-		manual				Urban			Chlorpyrifos	Occupational
39		m		IMS-		unemployed				Urban			Chlorpyrifos	Occupational
37		m		IMS-		unemployed				Urban			Malathion	Occupational
41		m		IMS-		manual				Urban			Chlorpyrifos	Occupational
38		m		IMS-		unemployed				Urban			Malathion	Occupational
42		m		IMS-		unemployed				Rural			Dimethoate	Occupational
37		m		IMS-		unemployed				Rural			Chlorpyrifos	Occupational
37		f		IMS-		unemployed				Rural			Malathion	Accidental
41		f		IMS-		unemployed				Rural			Chlorpyrifos	Accidental
38		f		IMS-		manual				Rural			Malathion	Accidental
23		f		IMS-		manual				urban			Chlorpyrifos	Accidental

PLAGARISM

10% Overall Similarity

The combined total of all matches, including overlapping sources, for each database.

Filtered from the Report

- Bibliography
- Quoted Text
- Small Matches (less than 10 words)

Match Groups

93 Not Cited or Quoted 9%
Matches with neither in-text citation nor quotation marks

3 Missing Quotations 0%
Matches that are still very similar to source material

0 Missing Citation 0%
Matches that have quotation marks, but no in-text citation

0 Cited and Quoted 0%
Matches with in-text citation present, but no quotation marks

Top Sources

7% Internet sources

7% Publications

0% Submitted works (Student Papers)

Integrity Flags

0 Integrity Flags for Review

No suspicious text manipulations found.

Our system's algorithms look deeply at a document for any inconsistencies that would set it apart from a normal submission. If we notice something strange, we flag it for you to review.

A Flag is not necessarily an indicator of a problem. However, we'd recommend you focus your attention there for further review.