

COMPARISON OF MODIFIED APACHE II, MEES, AND GLASGOW COMA SCORES IN MORTALITY PREDICTION OF PRESCHOOL CHILDREN WITH ACUTE POISONING-INDUCED COMA

Marwa A. Hasb Elnabi¹, Sarah A. Eweda², Alaa Essam Mahmoud²

¹Forensic Medicine and Clinical Toxicology Department, Faculty of Medicine, Sohag University, Egypt

²Forensic Medicine and Clinical Toxicology Department, Faculty of Medicine, Ain Shams University, Egypt

ABSTRACT

Background: Acute poisoning in children is regarded as a severe problem that frequently results in morbidity and death worldwide. Toxic coma is a potentially fatal illness, representing a great challenge for toxicologists. **Aim of the work:** The study aimed to describe the pattern and outcome of acute poisoning-induced coma among preschool children, as well as to compare modified Acute Physiology and Chronic Health Evaluation II (modified APACHE II), the Mainz Emergency Evaluation System (MEES), and the Glasgow Coma Scale (GCS) in mortality prediction. **Patients and methods:** A retrospective cross-sectional study was conducted on preschool children with acute poisoning-induced coma admitted to the ICU of PCC-ASUH from June 2022 to December 2023. **Results:** The study included seventy-four patients, 11 of whom were non-survivors. Most of the patients were male and two years old. Cannabis, clozapine, and hydrocarbons were the most frequent toxic agents inducing coma in preschool children. The mortality rate was 14.9%. The best cut-off points for predicting mortality in modified APACHE II, MEES, and GCS scores were > 9 , ≤ 18 , and ≤ 9 , with specificities of 90.4%, 93.6%, and 96.8%, respectively, and sensitivities of 100%. The MEES and GCS scores had the highest AUC (0.986), followed by the modified APACHE II (0.978). There was no statistically significant difference among the scores' AUCs. **Conclusion:** The modified APACHE II, MEES, and GCS scores were significant predictors of mortality in preschool children with acute poisoning-induced coma. GCS is easier to apply than other scores and is recommended for use.

Keywords: *The Mainz Emergency Evaluation System, Children, Toxic, Intensive care unit, Poison control center.*

Corresponding author: Dr. Marwa A. Hasb Elnabi

Email: Marwa_hafez@med.sohag.edu.eg

ORCID: 0000-0001-7373-330X

INTRODUCTION

Preschool age years range from birth to less than six years old and do not include full-time schooling. Children under the age of six are the future of any country and valuable resources for the sustained development of human society (Mohammed *et al.*, 2021).

Acute poisoning in children is a major public health issue around the world, and it is considered one of the top causes of accidental deaths. It is also a significant issue in developing countries, where it constitutes a prevalent reason for presentation and admission to emergency departments (Farag *et al.*, 2020).

Unconscious patients represent a diagnosis challenge in emergencies, but investigations into their features are few. Intoxication was

found to be the most frequent fundamental reason (Forsberg *et al.*, 2009).

Toxic substances can induce coma by directly affecting cells in the brain or by causing secondary disturbances, which may indirectly affect the functioning of the ascending reticular activating system (Young, 2009).

A coma induced by intoxication is a neurological emergency that necessitates immediate evaluation and treatment, particularly within the first hour. Specialized protocols and antidotes are needed (Buylaert, 2000).

Due to the scarcity of ICU beds, it is essential to understand the risk variables that can separate intoxicated patients into distinct survival groups. As a tool for triage and ICU quality monitoring, several scoring systems have been used (Eizadi-Mood *et al.*, 2011).

The Glasgow Coma Scale (GCS) scoring has been utilized to evaluate the outcome and recovery of individuals admitted to an ICU after a drug overdose. It has also been used to assess the mental state of poisoned individuals, the intubation needs of patients, and to predict acute and delayed intoxication outcomes (*Mohammed and Gawesh, 2019*).

The modified Acute Physiology and Chronic Health Evaluation II (modified APACHE II) score was utilized to evaluate critical ICU cases and has been used for patients with organophosphate poisoning with high sensitivity and specificity (*Yu et al., 2012; Dorooshi et al., 2023*).

The Mainz Emergency Evaluation Score (MEES) demonstrated excellent mortality prediction in seriously intoxicated individuals who required tracheal intubation and good outcome prediction in patients with mixed drug poisoning-inducing coma (*Eizadi-Mood et al., 2011; Seçgin and Fýrat, 2011*).

However, no previous studies compared the accuracy of different scoring systems in mortality prediction of preschool children with acute poisoning-induced coma.

THE AIM OF THE WORK

This study aimed to describe the pattern and outcome of acute poisoning-induced coma among preschool children, as well as to compare modified APACHE II, MEES, and GCS scores in mortality prediction.

PATIENTS AND METHODS

Study design and setting

This study was a retrospective observational cross sectional study.

Inclusion criteria: This study was conducted on all preschool aged children (ages 1 to 5) of both sex diagnosed with acute poisoning-induced coma and admitted to the ICU of the Poison Control Center of Ain Shams University Hospitals (PCC-ASUH) during the period from June 2022 to December 2023.

Exclusion criteria: Individuals who were already experiencing cardiac, neurological, or mental disorders, or who had undergone any pre-consultation treatment, as well as those whose coma was caused by pathological, metabolic, or traumatic reasons upon arrival. Any patient sheet with incomplete medical records that preclude accurate calculation of

the modified APACHE II, MEES, or GCS was also excluded.

The cases were divided into two groups based on their outcomes: the survivors group and the non-survivors group.

Ethical Considerations: The general director of the PCC-ASUH gave his official agreement. This study received approval by the Research Ethics Committee of the Faculty of Medicine at Ain Shams University (**approval number FMASU R52/2024**). Regarding the informed consent, it was waived because data were collected from medical records. All data was saved anonymously in order to maintain confidentiality. The information gathered was exclusively used for the purposes of the study.

Sample Size

The sample size was calculated using Power Analysis and Sample Size software version 15 (PASS 15), setting power at 80% and alpha error at 0.05. After reviewing previous study results (*Mohammed and Gawesh, 2019*), a sample size of at least 62 preschool children presented with toxic coma and admitted to the ICU is sufficient to achieve the work objective.

Data collection

Information was gathered from the sheets and computerized database of the patients. The data collected from the sheet of each patient comprised sociodemographic information (age, sex, and residence), toxicological information (causative agents, route of exposure, and manner of intoxication), delay time, length of hospital stays, and grades of coma by Reed's coma scale, as well as investigations, treatment, and outcome.

On admission, the GCS score was recorded for all participating patients. The pediatric modified GCS was utilized for children <2 years old and the standard GCS for those ≥2 years old. Modified APACHE II, and MEES scores were calculated within the first 24 hours of ICU admission.

The GCS score is determined by combining the scores of 3 components: eye opening (range=1–4), verbal response (range=1–5), and motor response (range=1–6). Because of the requirement for verbal interaction, clinicians cannot utilize the standard GCS scale to accurately evaluate preverbal

children. As a result, the pediatric GCS scale is a modified GCS scale designed to be used with preverbal children. A total GCS score of 3 indicates a deep coma or death, whereas a score of 15 indicates a fully conscious individual (*Borgialli et al., 2016; Mansour et al., 2018*).

The modified APACHE II was calculated using five physiological parameters: mean arterial pressure, temperature, respiratory rate, and heart rate, and each parameter was graded on a scale of 0 to 4, with 0 indicating normal and 4 indicating the most aberrant. Finally, the GCS score (15-GCS) was included. These numbers were put together with a mark that took the patient's age and chronic health conditions into account. The modified APACHE II can be estimated excluding biochemical parameters (arterial oxygen tension, arterial pH, serum sodium, serum potassium, serum creatinine, hematocrit, white blood cell count) that are generally utilized in the APACHE II Score (*Eizadi Mood et al., 2011*).

The MEES score is a descriptive scoring method that comprises GCS, heart rate, respiratory rate, systolic blood pressure, arterial oxygen saturation, ECG and pain (*Seçgin and Fýrat, 2011*).

STATISTICAL ANALYSIS

Data were collected, revised, coded, and entered into the Statistical Package for Social Science (IBM SPSS) version 27. The quantitative data were presented as mean, standard deviations, median, and interquartile range based on distribution. Also, qualitative data were shown as numbers and percentages. The chi-square test was used to compare qualitative data between groups. For quantitative data with a parametric distribution, the independent t-test was used, and for non-parametric data, the Mann-Whitney test. Receiver Operating Characteristic (ROC) curve was utilized to evaluate the best cut-off point with its sensitivity, specificity, positive predictive value, negative predictive value, and area under curve (AUC). The AUC was determined as follows: excellent (0.9–1), good (0.8–0.9), fair (0.7–0.8), poor (0.6–0.7), and fail (0.5–0.6) (*Jessen and Menard 1996*). Pairwise comparisons of the AUCs of the

scores under study were carried out using the method outlined by *DeLong et al. (1988)*. P-values less than 0.05 and 0.001 were considered significant and highly significant, respectively.

RESULTS

During the study period, seventy-four preschool children with acute poisoning-induced coma admitted to the ICU of PCC-ASUH were included in the study based on inclusion and exclusion criteria.

The patients were classified based on their final outcome into 63 survivors and 11 non-survivors with the mortality rate accounting for approximately 14.9%. In this study, most of the patients were two years old. The majority of cases was male and came from the Cairo governorate (66.2%). The most prevalent exposure route was oral (97.3%), the manner of intoxication was accidental (100%), and the majority of patients had a time delay within 3 hours.

No significant difference was found between survivors and non-survivors regarding sociodemographic data, route of exposure, manner of intoxication, or delay time. The most common toxic substances that induce coma in preschool children during the study were cannabis, representing 32.4% of patients, followed by clozapine (18.9%) and hydrocarbons (12.2%). Hydrocarbons and paraphenylene diamine were significant risk factors, as shown in **table (1)**.

Table (2) illustrates that heart rate was significantly higher among non-survivors compared to survivors, while SBP and DBP were significantly lower in non-survivors compared to survivors. No significant difference was detected between both groups regarding temperature or respiratory rate.

Concerning the consciousness level, it was assessed and graded according to Reed's coma scale as well as the GCS. It was found that grade I coma was the most frequent (79.7%), followed by grade II (9.5%). The majority of non-survivors were coma IV (45.5%), while most survivors were coma I (93.7%). Regarding GCS, the majority of cases (79.7%) had a GCS of 9 to 12. Both scales showed significant differences between survivors and non-survivors, as shown in **table (3)**.

Table (4) illustrates that non-survivors had significantly lower pH and higher PCo₂ compared to survivors. Although non-survivors had lower mean HCO₃ levels than survivors, the difference was insignificant.

No significant difference was observed regarding serum sodium and serum creatinine. Both potassium level and blood urea nitrogen (BUN) were significantly lower among non-survivors compared to survivors, while mean random blood sugar (RBS) was significantly higher among non-survivors compared to survivors, as shown in **table (5)**.

The majority of studied patients had a median hospital stay of 2 days, with no significant difference detected between survivors and non-survivors.

The majority of non-survivors needed mechanical ventilation, vasopressor infusion therapy, and the use of IV NaHco₃, and this was statistically significant (**Table 6**).

Non-survivors had a significantly higher modified APACHE II score than survivors. While non-survivors had significantly lower MEES score and GCS score compared to survivors, as shown in **table (7)**.

Table (8) and Figure (1) illustrate the ROC curve analysis for mortality prediction based on the three studied scores. All of the scores studied had an AUC of greater than 0.9, showing that they are excellent predictors of mortality in preschool children with acute poisoning-induced coma. Both the MEES and GCS scores had the highest AUC (0.986), followed by the modified APACHE II (0.978). In a pairwise comparison of the AUCs for the three scores, there was no statistically significant difference (all p values >0.05). The best cut-off values for each score, along with their related sensitivities, specificities, PPV, and NPV, are illustrated in **table (8)**.

Table (1): Sociodemographic (age, sex and residence) and intoxication data (route, manner of intoxication and toxic agent) of the patients under the study.

Characteristics		Non-survivors N= 11	Survivors N= 63	Total N= 74	Test value	P-value
Age (year)	Median (IQR)	3 (2 – 4)	2 (2 – 4)	2 (2 – 4)	-1.135 MW	0.257
Sex	Male	5 (45.5%)	38 (60.3%)	43 (58.1%)	0.850 χ^2	0.357
	Female	6 (54.5%)	25 (39.7%)	31 (41.9%)		
Residence	Cairo	6 (54.5%)	43 (68.3%)	49 (66.2%)	3.634 χ^2	0.163
	Giza	3 (27.3%)	5 (7.9%)	8 (10.8%)		
	Others	2 (18.2%)	15 (23.8%)	17 (23.0%)		
Toxic agent	Cannabis	1 (9.1%)	23 (36.5%)	24 (32.4%)	3.212 χ^2	0.073
	Clozapine	0 (0.0%)	14 (22.2%)	14 (18.9%)	3.015 χ^2	0.082
	Hydrocarbons	4 (36.4%)	5 (7.9%)	9 (12.2%)	7.084 χ^2	0.007**
	Organophosphates and carbamates	2 (18.2%)	3 (4.8%)	5 (6.8%)	2.677 χ^2	0.101
	Neurazine	0 (0.0%)	4 (6.3%)	4 (5.4%)	0.738 χ^2	0.390
	Unknown	1 (9.1%)	2 (3.2%)	3 (4.1%)	0.843 χ^2	0.358
	Baclofen	0 (0.0%)	2 (3.2%)	2 (2.7%)	0.359 χ^2	0.549
	Carbamazepine	0 (0.0%)	2 (3.2%)	2 (2.7%)	0.359 χ^2	0.549
	Mixed poisons	0 (0.0%)	2 (3.2%)	2 (2.7%)	0.359 χ^2	0.549
	Paraphenylene diamine	2 (18.2%)	0 (0.0%)	2 (2.7%)	11.773 χ^2	0.000**
	Snake bite	1 (9.1%)	1 (1.6%)	2 (2.7%)	2.005 χ^2	0.156
	Tramadol	0 (0.0%)	2 (3.2%)	2 (2.7%)	0.359 χ^2	0.549
	Anti-Parkinson drugs	0 (0.0%)	1 (1.6%)	1 (1.4%)	0.177 χ^2	0.673
	Oral hypoglycemic	0 (0.0%)	1 (1.6%)	1 (1.4%)	0.177 χ^2	0.673
	Paracetamol	0 (0.0%)	1 (1.6%)	1 (1.4%)	0.177 χ^2	0.673
Delay time (hours)	Median (IQR)	2 (1 – 7)	3 (2 – 7)	3 (2 – 7)	-0.731MW	0.465
Manner of intoxication	Accidental	11 (100%)	63 (100%)	74 (100.0%)	–	–
Route of exposure	Oral	10 (90.9%)	62 (98.4%)	72 (97.3%)	2.005 χ^2	0.157
	Bite or Sting	1 (9.1%)	1 (1.6%)	2 (2.7%)		

P-value < 0.01: highly significant (**). N: Number . IQR: interquartile range. χ^2 : Chi- Square test. MW: Mann-Whitney test.

Table (2): Vital signs of survivors and non-survivors preschool children with acute poisoning-induced coma.

Vital signs		Non-survivors N= 11	Survivors N= 63	Total N= 74	Test value	P-value
Pulse (Beats/min)	Mean \pm SD	143.27 \pm 30.54	117.54 \pm 26.73	121.36 \pm 28.63	2.885 t	0.005**
	Range	80 – 196	37 – 198	37 – 198		
SBP (mmHg)	Mean \pm SD	85.45 \pm 15.08	99.84 \pm 11.14	97.7 \pm 12.77	-3.741 t	0.000**
	Range	60 – 100	70 – 150	60 – 150		
DBP (mmHg)	Mean \pm SD	53.64 \pm 13.62	60.95 \pm 9.79	59.86 \pm 10.66	-2.151 t	0.035*
	Range	30 – 70	30 – 90	30 – 90		
Temperature °C	Mean \pm SD	37.29 \pm 0.9	37.21 \pm 0.43	37.22 \pm 0.52	0.485 t	0.629
	Range	36 – 39	36.5 – 38.5	36 – 39		
Respiratory rate (breaths /min)	Median (IQR)	37 (4 – 40)	26 (24 – 30)	26(24 – 34)	-1.255 MW	0.209
	Range	3 – 60	18 – 60	3 – 60		

P <0.05: significant (*) *P* <0.001: highly significant (**) *N*: Number. *SBP*: Systolic blood pressure. *DBP*: Diastolic blood pressure. *SD*: Standard deviation. *IQR*: interquartile range. *t*: Independent *t*- test. *MW*: Mann-Whitney test.

Table (3): Coma assessed by Reed's Coma Scale and Glasgow coma Scale of the patients in the study.

		Non-survivors N= 11	Survivors N= 63	Total	Test value	P-value
Reed's coma scale	Coma I	0 (0.0%)	59 (93.7%)	59 (79.7%)	55.186 χ^2	0.000**
	Coma II	4 (36.4%)	3 (4.8%)	7 (9.5%)		
	Coma III	2 (18.2%)	1 (1.6%)	3 (4.1%)		
	Coma IV	5 (45.5%)	0 (0.0%)	5 (6.8%)		
GCS	≤ 8	5 (45.5%)	1 (1.6%)	6 (8.1%)	24.825 χ^2	0.000**
	9 - 12	6 (54.5%)	53 (84.1%)	59 (79.7%)		
	> 12	0 (0.0%)	9 (14.3%)	9 (12.2%)		

P <0.001: highly significant (**). *N*: Number. χ^2 : Chi- Square test.

Table (4): Analysis of arterial blood gases in survivors and non-survivors preschool children with acute poisoning-induced coma.

Arterial blood gases		Non-survivors N= 11	Survivors N= 63	Total N= 74	Test value	P-value
Acid base status	Normal	0 (0.0%)	6 (9.5%)	6 (8.1%)	14.877 χ^2	0.005**
	Respiratory acidosis	2 (18.2%)	11 (17.5%)	13 (17.6%)		
	Respratory alkalosis	0 (0.0%)	3 (4.8%)	3 (4.1%)		
	Metabolic acidosis	5 (45.5%)	41 (65.1%)	46 (62.2%)		
	Mixed acidosis	4 (36.4%)	2 (3.2%)	6 (8.1%)		
PH	Mean \pm SD	7.18 \pm 0.14	7.32 \pm 0.08	7.3 \pm 0.1	-4.327 t	0.000**
	Range	6.9 – 7.36	7 – 7.46	6.9 – 7.46		
PCO2 (mmHg)	Mean \pm SD	53.11 \pm 17.78	39.38 \pm 9.24	41.42 \pm 11.83	3.880 t	0.000**
	Range	22.9 – 89	22 – 76	22 – 89		
HCO3 (mEq/L)	Mean \pm SD	23.25 \pm 2.95	24.52 \pm 4.03	24.33 \pm 3.89	-1.002 t	0.320
	Range	20 – 29.3	9.5 – 29.9	9.5 – 29.9		

P <0.001: highly significant (**). *N*: Number. χ^2 : Chi- Square test. *t*: Independent *t*- test.

Table (5): Laboratory investigations of survivors and non-survivors preschool children with acute poisoning-induced coma.

		Non-survivors N= 11	Survivors N= 63	Total N= 74	Test value	P-value
Sodium (mEq/L)	Mean \pm SD	236.64 \pm 5.8	237.02 \pm 4.96	236.96 \pm 5.05	-0.228 t	0.820
	Range	225 – 242	220 – 253	220 – 253		
Potassium (mEq/L)	Mean \pm SD	3.75 \pm 0.47	4.1 \pm 0.36	4.04 \pm 0.4	-2.731 t	0.008**
	Range	3.2 – 4.7	3.3 – 5.3	3.2 – 5.3		
RBS (mg/dl)	Mean \pm SD	288.64 \pm 97.41	190.03 \pm 66.07	204.69 \pm 79.08	4.235 t	0.000**
	Range	220 – 540	77 – 350	77 – 540		
BUN (mg/dl)	Mean \pm SD	9 \pm 1.48	11.17 \pm 2.47	10.85 \pm 2.46	-2.827 t	0.006**
	Range	8 – 12	8 – 24	8 – 24		
Creatinine (mg/dl)	Mean \pm SD	0.43 \pm 0.15	0.47 \pm 0.18	0.46 \pm 0.17	-0.689 t	0.493
	Range	0.2 – 0.7	0.2 – 0.9	0.2 – 0.9		

P-value < 0.01: Highly significant (**). RBS: Random blood sugar BUN: Blood urea nitrogen
 χ^2 : Chi-Square test. t: Independent t-test N: Number SD: Standard deviation

Table (6): Hospital stay duration, management of preschool children with acute poisoning-induced coma.

		Non-survivors N= 11	Survivors N= 63	Total N= 74	Test value	P-value
Hospital stay(days)	Median (IQR)	2 (1 – 4)	2 (1 – 2)	2 (1 – 3)	-1.329MW	0.184
Decontamination	None	10 (90.9%)	59 (93.7%)	69 (93.2%)	0.507 χ^2	0.776
	GL	1 (9.1%)	3 (4.8%)	4 (5.4%)		
	AC	0 (0.0%)	1 (1.6%)	1 (1.4%)		
Antidotes	None	9 (81.8%)	57 (90.5%)	66 (89.2%)	4.679 χ^2	0.456
	N- Acetyl Cysteine	0 (0.0%)	1 (1.6%)	1 (1.4%)		
	Anti-venom	1 (9.1%)	1 (1.6%)	2 (2.7%)		
	Atropine and oximes	0 (0.0%)	2 (3.2%)	2 (2.7%)		
	Naloxone	0 (0.0%)	1 (1.6%)	1 (1.4%)		
	Atropine	1 (9.1%)	1 (1.6%)	2 (2.7%)		
Enhanced elimination	None	11 (100%)	62 (98.4%)	73 (98.6%)	0.177 χ^2	0.674
	MDAC	0 (0.0%)	1 (1.6%)	1 (1.4%)		
	Dialysis	0(0.0%)	0 (0.0%)	0 (0.0%)		
Supportive treatment	I.V fluids	11 (100%)	63 (100%)	74 (100.0%)	–	–
	Mechanical ventilation	11 (100%)	3 (4.8%)	14 (18.9%)	55.374 χ^2	0.000**
	Dopamine	11 (100%)	0 (0.0%)	11 (14.9%)	74.000 χ^2	0.000**
	NaHCO ₃	10 (90.9%)	1 (1.6%)	11 (14.9%)	59.040 χ^2	0.000**

P-value < 0.01: highly significant (**). χ^2 : Chi-Square test. MW: Mann-Whitney test. IQR: interquartile range.

Table (7): Modified APACHE II, MEES, and GCS scores of survivors and non-survivors preschool children with acute poisoning-induced coma.

Score		Non-survivors N = 11	Survivors N = 63	Total N = 74	Test value	P-value
Modified APACHE II	Mean \pm SD	14.73 \pm 4.36	6.32 \pm 2.37	7.57 \pm 4.06	9.399 t	0.000**
	Range	10 – 23	3 – 14	3 – 23		
MEES	Mean \pm SD	16 \pm 1.67	22.17 \pm 1.96	21.26 \pm 2.92	-9.848 t	0.000**
	Range	13 – 18	17 – 26	13 – 26		
GCS	Mean \pm SD	7.09 \pm 2.7	11.94 \pm 0.86	11.22 \pm 2.15	-11.551 t	0.000**
	Range	3 – 9	8 – 13	3 – 13		

P < 0.001: highly significant (**). N: Number. GCS: Glasgow Coma Scale APACHE II: Acute Physiology and Chronic Health Evaluation II MEES: The Mainz Emergency Evaluation Score t: Independent t-test.

Table (8): Comparison of the studied scores for mortality prediction using ROC curve analysis.

	Modified APACHE II	MEES	GCS
AUC (95% CI)	0.978 (0.913 to 0.998)	0.986 (0.925 to 1.000)	0.986 (0.927 to 1.000)
P	<0.001**	<0.001**	<0.001**
Cut-off point	>9	≤18	≤ 9
Sensitivity	100	100	100
Specificity	90.48	93.65	96.83
PPV	64.7	73.3	84.6
NPV	100	100	100
P value from pairwise comparisons of AUCs			
Modified APACHE II		0.218	0.472
MEES	0.218		0.941
GCS	0.472	0.941	

AUC: Area under Curve. PPV: Positive Predictive Value. NPV: Negative Predictive Value. CI: confidence interval
 GCS: Glasgow Coma Scale APACHE II: Acute Physiology and Chronic Health Evaluation II
 MEES: The Mainz Emergency Evaluation Score P <0.001: highly significant (**)

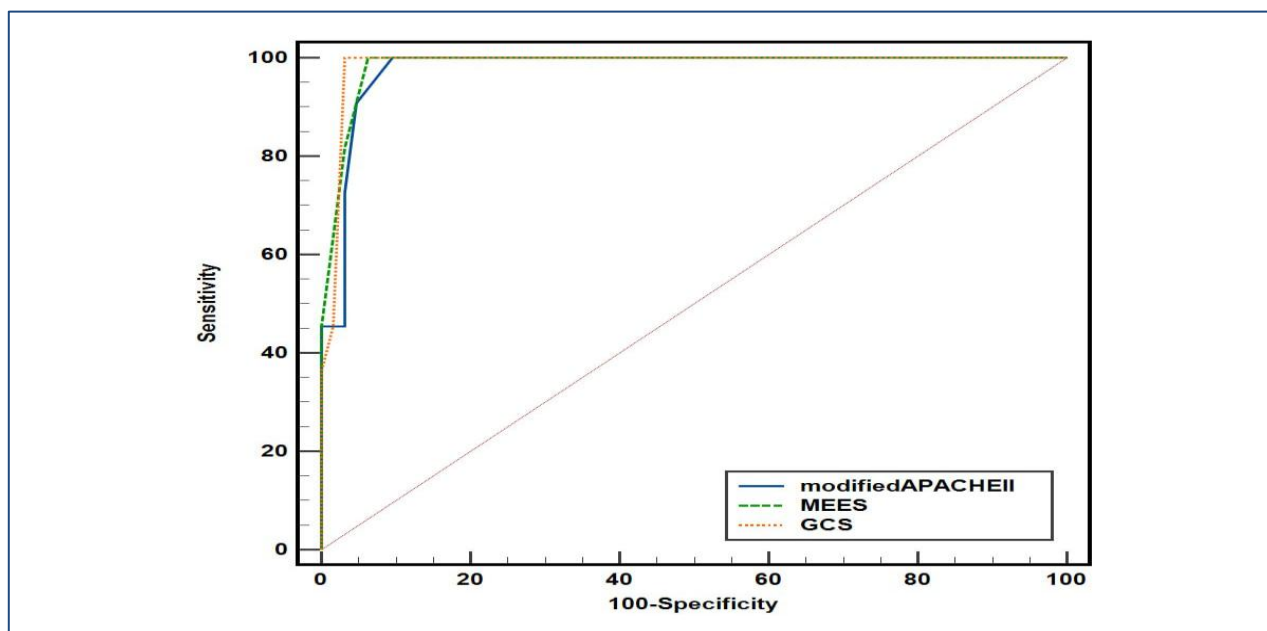


Figure (1): ROC curves for mortality prediction based on modified APACHE II, MEES, and GCS scores. The best cut-off points for predicting mortality for modified APACHE II, MEES, and GCS scores were > 9, ≤ 18, and ≤ 9, with specificities of 90.4%, 93.6%, and 96.8%, respectively, and sensitivities of 100%. The MEES and GCS scores had the highest AUC (0.986), followed by the modified APACHE II (0.978).

DISCUSSION

Comatose individuals are at increased risk for morbidity and death; a prompt and comprehensive diagnostic work-up to identify and perhaps cure the reason for their condition is imperative (David and Greer, 2013).

In the current study, there were 74 preschool children with acute poisoning-induced coma admitted to the ICU. The death rate was 14.9%.

This finding was in accordance with Moawad *et al.* (2015); Mohamed and Gawesh (2019); and Panda *et al.* (2015), where the death rate was 14.2%, 12%, and 15%, respectively, in comatose individuals.

On the contrary, Forsberg *et al.* (2009) reported that toxic coma was responsible for 2.8% of deaths. Sweilum and Kandeel (2022) revealed that the death rate of toxic coma was 6.4%, and the variation in mortality rates could be attributed to disparities in the

causative hazardous substances and the time it takes to get to the hospital.

The sociodemographic features of this study were similar to those in prior investigations. Male cases outweighed female cases, with those aged 2 years being the most affected. The most common route of exposure was oral; the manner of intoxication was accidental, and the majority of patients had a time delay within 3 hours.

This result is in harmony with that of *Zanaty and Girgis (2010)*; *Mohamed and Gawesh (2019)*; *Sweilum and Kandeel (2022)*, who found that the majority of patients were male. Also, *Mohamed and Gawesh (2019)* and *Moawad et al. (2015)* reported that the route of exposure was ingestion in most cases.

Ninety percent of intoxication cases involving children under the age of five are admitted, peaking at two years old, and are more common in lower socioeconomic groups. The reason for this is that parents may not be aware of harmful agents. One of the most dangerous illnesses and deaths that occur in children is poisoning, mainly from hazardous substances that are kept around the house. The interaction of the substance, the child, and the family setting results in intoxication (*Mohammed et al., 2021*).

Children under the age of five were involved in 90% of accidental poisoning cases. Intoxication in children is always the result of negligence in keeping toxic materials within their reach or insufficient supervision (*El Guindi, 2016*).

This finding was nearly the same as that recognized by *Mohamed and Gawesh (2019)*, who reported that the majority of cases arrived at the hospital between two and six hours (76%).

This work differs from earlier research on the poisonous substance that causes coma. The most prevalent hazardous substance that caused coma in this work was cannabis, followed by clozapine and hydrocarbons.

However, in two additional studies (*Talaie et al., 2007*; *Mousavi et al., 2015*), opioids were the most common kind of intoxication that caused coma. *Mohamed and Gawesh (2019)* reported that the most frequent toxic substance causing coma was

organophosphorus poisoning, followed by carbamazepine, then tramadol. According to *Dadpour et al. (2017)*, neuropsychiatric medication poisoning was the most common agent, with alcohol coming in second. According to a 2012 Swedish study, ethanol by itself, sedative-hypnotics, and finally ethanol in conjunction with sedative-hypnotics were the causes of toxic coma (*Forsberg et al., 2009*). *Sweilum and Kandeel (2022)* reported that drug overdose is the most frequent causative toxic substance, followed by insecticides.

The Fund for Drug Control and Treatment of Addiction's statistics show that cannabis is the most often abused substance in Egypt. Increased accessibility for kids is a result of widespread cannabis use (*Odejide and Morakinyo, 2016*).

Mohammed et al. (2021) reported that out of the 248 acutely cannabis-intoxicated children (younger than 18 years old) presented to PCC-ASUH throughout the examined duration (2019), 223 (89.9%) were preschool children (younger than 6 years).

Kerosene ingestion in children has a major impact on public health, mainly in children below the age of six. Hypoxemia on arrival and a higher frequency of secondary pneumonia are some of the poor prognostic factors described in patients with hydrocarbon poisoning (*Jayashree et al., 2006*; *Kumar and Parvathy, 2012*).

In terms of vital signs, the present study found that, in comparison to survivors, non-survivors had a significantly greater heart rate and significantly lower blood pressure.

Jayashree and Singh (2011) reported hypotension at admission as the most significant predictor of death in children admitted to the ICU with acute poisoning. *Yu et al. (2012)* stated that patients with vital signs of extreme value had a worse prognosis than others.

Reed's coma grade and death were shown to be significantly correlated in the current work, with grade IV patients having the greatest fatality rate (45.5%).

This was in accordance with *Hassanian-Moghaddam et al. (2007)* and *Mohamed and Gawesh (2019)*, who found that the grade IV death rate was greater at 34% and 66.7%,

respectively. Additionally, according to **Chadha (2003)**, individuals in grade IV require advanced measures to prolong life because these coma grades have greater death rates. Persons in grade III need intubation and admission in the ICU.

In addition, **Sweilum and Kandeel (2022)** reported that all fatalities occurred among patients with coma grades III and IV.

In the current study, non-survivors had significantly lower pH and higher P_{CO2} compared to survivors.

The same finding was reported by **Hua et al. (2017)**, who indicated that death was linked to a lower mean pH and a greater mean PaCO₂. Also, **Mohamed and Gawesh (2019)** reported that significant correlations were found between acidosis, respiratory failure, and fatality.

On the other hand, **Sweilum and Kandeel (2022)** reported that low PCO₂ and HCO₃ were substantially linked to the deaths of toxic coma patients.

The present study revealed that a lower potassium level, a lower BUN, and hyperglycemia indicated a poor outcome.

This was similar to **Mohammed et al. (2021)**, where the severity of intoxication had an impact on random blood sugar levels. **Claudet et al. (2017)** found that a larger proportion of patients (76%) had increased random blood glucose levels, particularly those who were extremely agitated. This may be attributed to the related activation of the sympathetic nervous system, which causes an increase in random blood glucose levels in agitated patients.

This is in accordance with **Sinekalatha et al. (2019)**, who found that the occurrence of electrolytes and acid base abnormalities was significantly higher in non-survivors than survivors and explained that hypokalemia may be due to repeated vomiting in poisoned patients or to the direct effect of the toxin itself.

According to this study's treatment measures, a significant association was observed between mortality and the requirement for mechanical ventilation.

This was consistent with **Mohamed and Gawesh (2019)**, who revealed a significant relationship between the necessity for

intubation or mechanical ventilation and death.

In this study, non-survivors had a significantly lower GCS score than survivors. This is in accordance with **Budhathoki et al. (2009)**, who discovered that GCS < 8 was more related to death in intoxicated children.

On the contrary, **Mohamed and Gawesh (2019)** and **Kheirabadi et al. (2015)** reported that there was no significant relationship between the GCS and death.

To the authors' knowledge, this study is the first to compare the accuracy of the modified APACHE II score, MEES score, and GCS score as predictors of in-hospital mortality in preschool children with acute poisoning-induced coma.

In this study, ROC curve analysis to assess the predictors of mortality showed that the modified APACHE II score had an excellent AUC (0.978), 100% sensitivity, and 90.4% specificity at a cut-off value > 9.

These findings are consistent with those of **Eizadi-Mood et al. (2011)**, who found that the modified APACHE II score calculated at 24 hours had a good AUC (0.86), 100% sensitivity, and 61% specificity at cut-off point 10, providing more accurate outcome predictions for patients with mixed drug poisoning-induced coma.

In this study, the MEES and GCS scores demonstrated the highest AUC (0.986) at cut-off values ≤ 18, ≤ 9, with specificities of 93.6% and 96.8%, respectively, and 100% sensitivity.

This result is consistent with the findings of **Eizadi-Mood et al. (2011)**, who reported that the GCS assessed at 24 hours of admission was highly predictive of outcome, with an AUC of 0.90, 83.3% sensitivity, and 94.6% specificity at a cut-off value of ≤ 5. Additionally, the MEES at 24 hours showed 83.3% sensitivity, 73.4% specificity, and a good AUC of 0.80 at a cut-off point of ≤ 18 in patients with coma induced by mixed drug poisoning.

Also, **Seçgin and Fýrat (2011)** found that the MEES score accurately predicted death in severely intoxicated persons who required tracheal intubation, with an AUC of 0.920, 100% sensitivity, and 74% specificity at the cut-off point of 14.

Initial assessment of GCS may help the clinician to identify advanced grade of OP poisoning patients, which has been illustrated by *Akdur et al. (2010)*. GCS has been utilized for prediction of delayed neuropsychological sequels of carbon monoxide intoxication (*Ku et al., 2010*).

Given that there is no significant difference in the discriminatory power of the three scores, we can recommend using the GCS score because it is a simple and speedy score that can be easily applied in acute poisoning and other emergency circumstances. Additionally, it does not require extensive clinical or laboratory data. On the other hand, modified APACHE II and MEES scores involve several parameters, making them more complex, time-consuming to calculate, and less useful for rapid assessment.

CONCLUSION

Cannabis, clozapine, and hydrocarbons were the most frequent toxic substances inducing coma in preschool children. The mortality rate was 14.9%. Hydrocarbons and paraphenylene diamine were significant risk factors. The mortality was significantly associated with tachycardia, hypotension, a higher grade of Reed's coma scale, arterial blood gas abnormalities (acidosis, high PCO₂), a higher modified APACHE II score, a lower MEES score, a lower GCS score, and mechanical ventilation. The modified APACHE II, MEES, and GCS scores were significant predictors of mortality in preschool children with acute poisoning-induced coma. GCS is easier to apply than other scores and is recommended for use.

RECOMMENDATIONS

To confirm the current study's findings and assess the predictive value of these indices for unfavorable outcomes, multicenter studies with larger sample sizes are required.

Availability of data and materials: The corresponding author can provide the datasets created and/or analyzed during the current work upon reasonable request.

Declaration of interests: The authors have disclosed no relevant financial or non-financial interests.

Funding: The authors received no support from any organizations.

REFERENCES

1. **Akdur, O.; Durukan, P.; Ozkan, S. et al. (2010):** Poisoning severity score, Glasgow coma scale, corrected QT interval in acute organophosphate poisoning. *Hum. Exp. Toxicol.*,29:419-425.
2. **Borgialli, D.; Mahajan, P.; Hoyle, J. et al. (2016):** Performance of the Pediatric Glasgow Coma Scale Score in the Evaluation of Children with Blunt Head Trauma. *Acad. Emerg. Med.*,23(8):878-884. DOI: 10.1111/acem.13014.
3. **Budhathoki, S.; Poudel, P.; Shah, D. et al. (2009):** Clinical profile and outcome of children presenting with poisoning or intoxication: A hospital based study. *Nepal Med. Coll. J.*,11:170-175.
4. **Buylaert, W. (2000):** Coma induced by intoxication. *Acta. Neurol. Belg.*,100(4): 221-224.
5. **Chadha, I. (2003):** Poisoning. *Ind. J. Anesth.*, 47: 402-411.
6. **Claudet, I.; Le Breton, M.; Brehin, C. et al. (2017):** A 10-year review of cannabis exposure in children under 3-years of age: do we need a more global approach?. *Eur. J. Pediatr.*,176(4):553-556.
7. **Dadpour, B.; Tajoddini, S.; Shaarbaf, E. et al (2017):** Role of serum creatinine phosphokinase in outcome prediction of intoxicated patients; a Brief Report. *Emerg.*,5(1): 1-4.
8. **David, M. and Greer, M. (2013):** Clinical evaluation of coma and brain death. *Neurocrit. Care Soc. Prac. Update*, 30: 359-364.
9. **DeLong, E.; DeLong, D. and Clarke-Pearson, D. (1988):** Comparing the areas under two or more correlated receiver operating characteristic curves: a nonparametric approach. *Biometr.*,44(3):837-845.
10. **Dorooshi, G.; Samsamshariat, S.; Gheshlaghi, F. et al. (2023):** Comparing sequential organ failure assessment score, acute physiology and chronic health evaluation II, modified acute physiology and chronic health evaluation II, simplified acute physiology score II and poisoning severity score for outcome prediction of pesticide poisoned patients admitted to the intensive care unit. *J. Res. Pharm. Pract.*,12:49-57.
11. **Eizadi-Mood, N.; Sabzghabae, A. and Dehkordi, K. (2011):** Applicability of different scoring systems in outcome prediction of patients with mixed drug poisoning-induced coma. *Ind. J.*

- Anesth.*,55(6):599-604. DOI:10.4103/0019-5049.90616
12. **El Guindi, K. (2016):** Common poisoning in infancy and childhood, published thesis for master degree. *Ain Shams Univers.*,57-58.
 13. **Farag, A.; Said, E. and Fakher, H. (2020):** Patterns of Pediatric Acute Poisoning at Banha Poisoning Control Center, Egypt: One-Year Prospective Study. *Asia Pac. J. Med. Toxicol.*, 9(2):44-51.
 14. **Forsberg, S.; Höjer, J.; Enander, C. et al. (2009):** Coma and impaired consciousness in the emergency room: characteristics of poisoning versus other causes. *Emerg. Med. J.*, 26:100–102. DOI:10.1136/emj.2007.054536
 15. **Hassanian-Moghaddam, H.; Baghbanian, N.; and Kolahi, A. (2007):** Evaluating the factors accompanying coma in patients who admitted in poisoning ICU in Loghman Hakim Hospital. *Iran. J. Toxicol.*,1(2):56-63.
 16. **Hua, A.; Haight, S.; Hoffman, R. et al (2017):** Endotracheal Intubation after Acute Drug Overdoses: Incidence, Complications, and Risk Factors. *J. Emerg. Med.*, 52:59–65. doi:10.1016/j.jemermed.2016.07.114
 17. **Jayashree, M.; Singhi, S.; Gupta, A. (2006):** Predictors of outcome in children with hydrocarbon poisoning receiving intensive care. *Ind. Pediatr.*, 43: 715-719.
 18. **Jayashree, M. and Singhi, S. (2011):** Changing trends and predictors of outcome in patients with acute poisoning admitted to the intensive care. *J. Trop. Pediatr.*,57 (5): 340–346.
 19. **Jessen, H. and Menard, S. (1996):** Applied logistic regression analysis. *Stat.*, 45:534. DOI:10.2307/2988559
 20. **Kheirabadi, A.; Tabeshpour, J. and Afshari, R. (2015):** Comparison of three consciousness assessment scales in poisoned patients and recommendation of a new scale: AVPU Plus. *Asia Pac. J. Med. Toxicol.*,4:58-63. DOI:10.22038/APJMT.2015.5080
 21. **Ku, H.; Yang, K.; Lee, Y. et al. (2010):** Predictors of carbon monoxide poisoning-induced delayed neuropsychological sequel. *Gen. Hosp. Psychiat.*,32:310 -314.
 22. **Kumar, S. and Parvathy, V. (2012):** A study of accidental ingestion of hydrocarbons in children in a medical college hospital in central Kerala. *Pediatr. Rev.: Int. J. Pediatr. Res.*, 3(5): 297-302.
 23. **Mansour, G.; Aglan M.; Abd Elwahab M. (2018):** Acute methanol poisoning: prognostic factors and role of Glasgow Coma Scale. *Egypt. J. Forensic Sci. App. Toxicol.*, 18 (4):41-51.
 24. **Moawad, A.; Abd El-Salam, M.; Ali, A. et al. (2015):** Toxic Coma Incidence at Kasr-Alainy National Poison Center [master's thesis]. *Cairo Univers.*, P (63-95).
 25. **Mohammed, A.; Osman H.; Azab G. (2021):** Evaluation of Acute Cannabis Intoxication in Pre- School Children Admitted to Poison Control Center–Ain Shams University Hospitals. *Ain Shams J. Forensic Med. and Clin. Toxicol.*,37: 16-25.
 26. **Mohammed, E. and Gawesh, E. (2019):** Acute poisoning induced coma: characteristics and predictive role of early creatine phosphokinase on its outcome. *Ain Shams J. Forensic Med. and Clin. Toxicol.*,32: 1-9.
 27. **Mohammed, F.; Sobhy, D. and Abd Elrahman B. (2021):** Mothers Perception regarding Poisoning among their Preschool Children. *J. Nurs. Sci. Benha Univers.*, 2(2):1-15. DOI:10.21608/jnsbu.2021.186169.
 28. **Mousavi, S.; Vahabzadeh, M.; Mahdizadeh, A. et al. (2015):** Rhabdomyolysis in 114 patients with acute poisonings. *J. Res. Med. Sci.*,20:239-243.
 29. **Odejide A. and Morakinyo J. (2016):** A community based study of patterns of psychoactive substance use among street children in a local government area of Nigeria. *Drug Alcohol Depend.*,71(2):109-116.
 30. **Panda, B.; Hansda, M.; Mishra, K. et al (2015):** Study of Poisoning Cases in an Indian Tertiary Care Teaching Hospital. *J. Ind. Acad. Forensic Med.*, 37:165-168. DOI: 10.5958/0974-0848.2015.00040.8
 31. **Seçgin, S. and Fýrat, B. (2011):** Comparison of the scoring systems for predicting mortality in intoxicated patients hospitalized to the ICU: a prospective observational study. *Erciyes Med. J.*,33(1):029-034.
 32. **Sinekalatha, J.; Narayanan, S. and Britto, R. (2019):** Electrolyte and acidbase disturbances in the critically ill patients: A retrospective case control study. *Int. Arch. Integ. Med.*,6(6): 1-8.
 33. **Sweilum, O. and Kandeel, F. (2022):** Characteristics of toxic coma and the role of Total antioxidant capacity (TAC) as a prognostic marker. *Egypt. J. Forensic Sci. App. Toxicol.*, 22(4):33-46. DOI:10.21608/ejfsat.2022.96491.1222.

34. **Talaie, H.; Pajouhmand, A.; Abdollahi, M. et al (2007):** Rhabdomyolysis among acute human poisoning cases. *Hum. Exp. Toxicol.*,26: 557-561. DOI:10.1177/0960327107078667
35. **Young, G. (2009):** Coma. *Ann. New York Acad. Sci.*, 1157: 32–47. DOI:10.1111/j.1749-6632.2009.04471.x
36. **Yu, J.; Weng, Y.; Chen, K. et al. (2012):** Triage vital signs predict in-hospital mortality among emergency department patients with acute poisoning: a case control study. *BMC Health Serv. Res.*, 12 (1): 1-8.
37. **Zanaty, A. and Girgis, N. (2010):** Toxic coma characteristics and predictors of outcome in cases admitted to Menoufia poisoning and addiction control center [MPCC]. *Ain Shams J. Forensic Med. Clin. Toxicol.*,15: 61-76.

مقارنة مقياس التسجيل اباتش الثانى المعدل ومقياس ماينز لتقييم الطوارئ ومقياس جلاسجو في التنبؤ بمعدلات الوفيات في مرضى الغيبوبة الناجمة عن التسمم الحاد لدى الأطفال في سن ما قبل المدرسة

مروة أحمد حسب النبي^١، سارة عاطف عويضة^٢، آلاء عصام محمود^٢
^١قسم الطب الشرعى والسموم الاكلينيكية، كلية الطب البشرى، جامعة سوهاج، مصر
^٢قسم الطب الشرعى والسموم الاكلينيكية، كلية الطب البشرى، جامعة عين شمس، مصر

الملخص العربى

المقدمة: يعتبر التسمم الحاد عند الأطفال مشكلة خطيرة تؤدي في كثير من الأحيان إلى المضاعفات المرضية والوفاة في جميع أنحاء العالم. كما ان الغيبوبة السامة قد تكون مميتة وتمثل تحديًا عظيمًا للمتخصصين في السموم.

الهدف: حيث هدفت هذه الدراسة الى وصف نمط ونتائج الغيبوبة الناجمة عن التسمم الحاد بين الأطفال في سن ما قبل المدرسة و مقارنة مقياس التسجيل اباتش الثانى المعدل ومقياس ماينز لتقييم الطوارئ و مقياس جلاسجو في التنبؤ بمعدلات الوفيات.

طريقة البحث: وقد تم اجراء هذه الدراسة المستعرضة الاسترجاعية على جميع الأطفال في سن ما قبل المدرسة من كلا الجنسين المصابين بالغيبوبة الناجمة عن التسمم الحاد والذين قد تم حجزهم في وحده العناية المركزة بمركز علاج التسمم بمستشفيات جامعة عين شمس خلال الفترة من يونيو ٢٠٢٢ الى ديسمبر ٢٠٢٣ .

النتائج: وقد اظهرت نتائج هذه الدراسة انه تم تسجيل اربعة وسبعون مريضاً في الدراسة، منهم ١١ مريض من غير الناجين. وقد كان اغلب المرضى من الذكور بعمر السنتين. وكان القنب والكلوزابين والهيدروكربونات هي العوامل السامة الاكثر شيوعاً التي تسببت في الغيبوبة عند الأطفال في سن ما قبل المدرسة. وكان معدل الوفيات في هذه الدراسة ٤.٩%. ان أفضل النقاط الفاصلة للتنبؤ بمعدلات الوفيات بالنسبة لمقياس التسجيل اباتش الثانى المعدل ومقياس ماينز لتقييم الطوارئ ومقياس جلاسجو هي $9 >$ و $18 \leq$ و $9 \leq$ بدقة مقدارها ٩٠.٤%، ٩٣.٦%، و ٩٦.٨% على التوالي، وبحساسية مقدارها ١٠٠%. وقد وجد ان كلا من مقياس ماينز لتقييم الطوارئ ومقياس جلاسجو قد سجلا اعلى معدل انحدار لوجيستي (٠.٩٨٦) يتبعهم مقياس اباتش الثانى المعدل (٠.٩٧٨). كما انه لم يكن هناك فارق ذو دلالة إحصائية بين الأنظمة الثلاثة من حيث قيمة الانحدار اللوجيستي لكل منهم.

الاستنتاج: وقد خلصت هذه الدراسة إلى أن مقياس التسجيل اباتش الثانى المعدل ومقياس ماينز لتقييم الطوارئ ومقياس جلاسجو من المنبئات الهامة لمعدلات الوفيات في مرضى الغيبوبة الناجمة عن التسمم الحاد بين الأطفال في سن ما قبل المدرسة. كما يعد مقياس جلاسجو الاسهل في التطبيق من الانظمة الاخرى لذ يوصى باستخدامه