CHARACTERISTICS OF TOXIC COMA AND THE ROLE OF TOTAL ANTIOXIDANT CAPACITY (TAC) AS A PROGNOSTIC MARKER

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ABSTRACT

Objectives: Coma is a state of loss of consciousness due to the disfunction of ascending reticular activating system. It is considered a life-threatening condition that needs immediate intervention. The body's total antioxidant capacity (TAC) is a biomarker that represents the amount of all endogenous antioxidants in the blood and body fluids. This study aims to analyze the characteristics of toxic coma and evaluate the predictive role of TAC analysis and other variables on the toxic coma outcome. **Methodology:** This prospective study was done on all patients diagnosed with toxic coma and admitted to MPCC of Menoufia university hospitals over one year of study. We recorded socio-demographic data (including age, sex, residence), causative toxic agent, and time passed from poison exposure to the hospital arrival, the manner of poisoning, duration of hospital stays, and outcome (Recovery or death). Investigations as arterial blood gases (pH, PO2 PCO2, HCO3) and serum TAC were measured. **Results:** The study found that the incidence of toxic coma was higher in the age group 15 - < 40 years, male gender, and delayed hospital arrivals. Drug overdose is the most common causative toxic agent, followed by insecticides, suicidal manner, longer duration of hospitalization, and residence in a rural area. Mortality was associated with male sex, delayed hospital arrival (> 4 hours), higher coma grades (Ⅲ& Ⅳ), long duration of hospital stay (> one week), abnormal ABG (acidosis, low PO_2, decreased HCO_3, low PCO_2). Factors that predispose the patients to poor outcomes and predict mortality are extended hospital stay decreased HCO3, delayed hospital arrival, and a low level of TAC. TAC can be used as a prognostic factor in toxic coma cases. **Conclusion**: TAC may be used as a prognostic marker in patients of toxic coma together with other predictive factors. **Recommendations:** Future studies on large-scale populations are needed to confirm the present study results.

Keywords: Toxic Coma, Mortality, Total antioxidant capacity, Predictive factors.

INTRODUCTION

Coma is a state of loss of consciousness due to dysfunction of the ascending reticular activating system (Young 2009). It is considered a life-threatening condition that needs immediate intervention (Stevens and Bhardwaj 2006)

Coma can originate from many different etiologies, such as toxic, metabolic, traumatic, and structural (Michelson and Ashwal 2004). Coma, due to poisonous causes, predominates other causes of coma. (Forsberg 2009).

A toxic coma represents a neurological emergency requiring rapid assessment and therapeutic action; this is especially important in the first hour. Specialized protocols with specific antidotes are required (Buylaert 2000).

Several grading systems for coma have been developed based on physiological state, spontaneous activity, and stimuli reactivity. Reed's classification of coma is one of the most straightforward grading systems (Teasdale and Jennett 1947) (Jennett and Bond 1975).

The imbalance between the production of

Reactive oxygen species (ROS) and antioxidant defense inside the human body are oxidative stress. (Wagner and Brath 2012).

The antioxidant defense system in the human body counteracts ROS and reactive nitrogen species (RNS); it consists of a mixture of various endogenous and exogenous chemicals that protect cellular targets from oxidative damage. (D.U. et al. 2009) The body's total antioxidant capacity (TAC) is the biomarker that represents the amount of all endogenous antioxidants in the blood and body fluids (Rani and Mythili 2014).

Antioxidant status has been measured using a variety of assays. But measuring the TAC is preferable, as it is a single test that measures all antioxidant activity (Koracevic 2001). TAC levels decreased in conditions associated with oxidative stress in many pathological and toxic conditions (Woodford and Whitehead 1998).

AIM OF THE STUDY:

This prospective study aims to analyze the prevalence of coma caused by acute poisoning among patients admitted to the Poison Control Center of Menoufia University Hospitals (MPCC) over one year of study (Mar 1, 2020, to the end of February 2021).

This study also aims to analyze the leading causes of toxic coma, the socio-demographic data, and coma grades according to Reed's classification. Also, the manner of poisoning, presentation time, hospital stay duration, arterial blood gases (ABG) changes, total antioxidant capacity level (TAC), and the patient's outcome. In addition, it aims to evaluate the predictive role of TAC analysis and other variables on the toxic coma outcome.

PATIENTS AND METHODS

This present study is a prospective study carried out at the Menoufia Poison Control Center (MPCC), Menoufia University Hospitals, Egypt, for one year (from Mar 1, 2020, to the end of February 2021).

Study design:

The study was done on all patients diagnosed with toxic coma and admitted to MPCC of Menoufia University Hospitals, Egypt, for over the year of study.

The Menoufia University Research Ethical Committee gave its approval to the study.

A complete history, clinical examination, and investigations were used to diagnose all the patients. We recorded the Socio-demographic data (including age, sex, residence), causative toxic agent; time passed from poison exposure to the hospital arrival, manner of poisoning, duration of hospital stays, and outcome (Recovery or death).

Each patient's guardian signed a consent form after discussing the aim of the work with him.

Investigations of Arterial blood gases (pH, PO_2, PCO_2, HCO_3) and toxicological analysis were done when needed, and serum TAC was measured using Koracevic, D. et al. technique (Koracevic et al. 2001).

The Reed scale, which has four degrees, was used to determine the extent of the coma: individuals in the first-degree coma had dysarthria, sensitivity to pain stimuli, and reflex reactions. Patients in a second-degree coma do not respond to verbal or painful stimulation, although they have reflex reactions. Patients with reduced reflexes and vital functions were classed as third-degree coma. Patients in the fourth degree of coma were non-responsive and unstable. (Behrman and Kliegman 2000; Stack and Dobbs 2008).

Exclusion criteria:

We exclude heavy smoking patients as it can affect TAC levels. In comparison to non-smokers, smokers had lower plasma TAC values (Goraca and Skibska 2005).

STATISTICAL ANALYSIS OF THE DATA:

The IBM SPSS software program version 20.0 was used to examine the data supplied to the computer (Armonk: IBM Corp). The Chi-square test was used to analyze group comparisons for

categorical variables (Fisher or Monte Carlo). The Mann-Whitney test was employed to compare two groups for not regularly distributed quantitative variables. Kruskal Wallis test was used to compare different groups for abnormally distributed quantitative variables. Regression is used to find the most influential/independent factor in death. The significance of the acquired results was determined at a 5% level of significance.

RESULTS

The total number of studied toxic coma patients who arrived at the Menoufia poisoning control center (MPCC) was 186. The mortality rate was 6.5% (12 patients) of all studied toxic coma patients (Figure 1).

The patients were divided into two groups based on their outcomes: group A (survivors) and group B (dead patients). Table 1 compared both studied groups regarding age, sex, causative toxic agents, manner of poisoning, grade of coma, duration of hospital stay, time of presentation, and ABG changes.

Half of the toxic coma patients (50 %) were between the ages of 15 and 40, with the age group 15 years following closely behind (35.5%). The age group 40- 60 years was 10.2% of patients. The lowest percentage was amongst the patients above 60 years (4.3%); most of the patients in groups A and B were in the age group 15-<40 years; There was no statistical difference in the outcome between the age groups. The total number of male patients was slightly more than that of female patients (54.3% Vs. 45.7%). Mortality was significantly higher in males (P-value 0.007) (Table 1).

Regarding the toxic coma causative toxic agents, drug overdose was the most common cause (67.7% of cases), insecticide exposure (15.6%), other causes such as Hashish ingestion, carbon monoxide (C.O.), snake bite, agrochemicals (dormex) and alcohol ingestion represented 4.3%, 3.8%, 3.2%, 0.5 and 0.5% respectively. The name of the causing hazardous substance was unknown to 4.3 percent of patients. There was no statistically significant difference between the two groups (A and B) regarding the causative toxic agent (Table 1). Regarding the drugs causing toxic coma, antipsychotics accounted for 38.09%; sedative-hypnotics were 19.05%, mixed drugs represented 17.46%, while antiepileptics, tricyclic antidepressants (TCA), muscle relaxants, antihistaminics, and tramadol represented 7.94%, 7.94%, 3.97%, 3.17%, and 2.38% respectively (Figure 2).

In almost half of the patients, toxic coma occurred suicidally (50.5%), 49% occurred accidentally, and only 0.5% occurred homicidally. The difference between both groups (A&B) regarding the mode of poisoning was statistically non-significant (Table 1).

Within 2-4 hours of toxin exposure, 52.2% of toxic coma patients arrived at MPCC. In surviving patients (group A), 52.9% arrived at MPCC within 2-4 hours of toxin exposure. While 58.3% of dead patients (group B) had a delayed presentation (> 4 hours) compared with group A (20.7%), there was a statistically significant difference (P- value 0.007) (Table 1). According to Reed's coma classification, nearly half of toxic coma patients (49%) were classified as grade I; grade II was represented by 36% of the total., while grades III and IV (all admitted to ICU) represented 11.8%, and 3.2% respectively. Coma grade I and II were significantly more in group A, while coma grade 3 and 4 was significantly higher in group B (Table 1).

Only 73.1% of the total toxic coma patients stayed less than two days in the hospital (73 percent of group A &75 percent of group B). The Majority of the patients who remained in the hospital for more than seven days died (3 out of 4), which is statistically highly significant (P 0.001). Regarding arterial blood gases (ABG), all patients in group B had a low pH (acidosis). In contrast, only 22.4% of patients had acidosis in group A. This difference between the two groups was statistically highly significant (P-value < 0.001). A low HCO_3 was represented in 75% of patients in group B, compared with only 13.2% in group A. This difference was statistically significant (P-value 0.001) between the two

groups. All patients in group B had low PO_2 (oxygen saturation), while only 14.9% of group A had low PO_2; this difference was statistically highly significant (P-value <0.001). All cases of group B showed a low PCO_2 level in their ABG compared with only 13.2% in group A, and this difference was statistically highly significant (P-value <0.001) (Table 1).

Table 2 shows the relationship between toxic coma grade (according to Reed's classification) and causative toxic agent, poisoning mode, and presentation time.

Regarding the relation between grades of coma and causative toxic agents, the Majority of coma grades I& II were caused by drug overdose (80.2% & 67.2%, respectively), and this difference was statistically significant. Insecticide poisoning was mainly presented with coma grade III (63.6%), and this difference was statistically significant. Carbon monoxide poisoning was mainly presented with coma grade II, and this difference was statistically significant. Half of coma grade IV (50%) was due to drug overdose.

Regarding the manner of poisoning, 52.7% of coma grade I occurred accidentally. Coma grade II was mainly caused accidentally and suicidally (49.3% each), grade III was caused mainly suicidally (68.2%). In comparison, grade IV occurred only accidentally and suicidally (50% each); these differences were statistically non-significant.

Half of the patients (50%) who presented with coma grade IV had a delayed arrival (>4 hours), and this was statistically significant (P-value 0.005).

All cases of carbon monoxide toxicity and unknown poison toxicity occurred in patients from rural areas; this was statistically significant (P-values 0.04 & 0.02, respectively) (Figure 3).

Table 3 shows the relationship between the total antioxidant capacity (TAC), coma grade, and causative toxic agents. TAC was lowest in coma grade IV compared with other coma grades, and this difference was statistically highly significant (P <0.001). The TAC showed a non-significant difference with the causative toxic agents (P 0.082). Figure 4 shows the relationship between the total antioxidant capacity (TAC) and the outcome. The median TAC of group A was 0.56 (0.06 –0.88), while it was 0.21 (0.02 –0.50) in group B. TAC in group B was statistically considerably lower (P-value 0.001).

By assessing the Multivariate regression analysis for the significant variables deduced from the univariate regression, it was found that duration of stay more than one week, a decrease of HCO_3, and a decrease of TAC are significantly independent markers for the bad outcome of toxic coma cases (table 4)

TABLE (1): COMPARISON BETWEEN THE TWO STUDIED TOXIC COMA GROUPS (GROUP A & GROUP B) TO DIFFERENT PARAMETERS.

	Total (n= 186)	Outcome		c^2	p
		Death (group B) (n= 12)	Survived (group A) (n= 174)		
Age					
<15	66 (35.5%)	2 (16.7%)	64 (36.8%)	1.984	^{FE}p=0.218
15 – 40	93 (50%)	7 (58.3%)	86 (49.4%)	0.356	0.551
40 – 60	19 (10.2%)	1 (8.3%)	18 (10.3%)	0.050	^{FE}p=1.000
>60	8 (4.3%)	2 (16.7%)	6 (3.4%)	2.765	^{FE}p=0.086
Sex					
Male	101 (54.3%)	11 (91.7%)	90 (51.7%)	7.217*	0.007*
Female	85 (45.7%)	1 (8.3%)	84 (48.3%)		
Toxic agent					
Drug	126 (67.7%)	5 (41.7%)	121 (69.5%)	3.991	^{FE}p=0.058
Insecticide	29 (15.6%)	3 (25%)	26 (14.9%)	0.863	^{FE}p=0.404
Hashish	8 (4.3%)	0 (0%)	8 (4.6%)	0.577	^{FE}p=1.000
Co	7 (3.8%)	0 (0%)	7 (4%)	0.502	^{FE}p=1.000
Snake	6 (3.2%)	0 (0%)	6 (3.4%)	0.428	^{FE}p=1.000
Unknown	8(4.3%)	2 (16.7%)	6 (3.4%)	4.765	^{FE}p=0.086
Dormex	1(0.5%)	1 (8.3%)	0 (0%)	14.578	^{FE}p=0.065
Alcohol	1(0.5%)	1 (8.3%)	0 (0%)	14.578	^{FE}p=0.065
Manner of poisoning					
Accidental	91 (48.9%)	4 (33.3%)	87 (50%)	1.248	0.264
Suicidal	94 (50.5%)	8 (66.7%)	86 (49.4%)	1.335	0.248
Homicidal	1 (0.5%)	0 (0%)	1 (0.6%)	0.069	^{FE}p=1.000
Time of presentation					
Within 2h	46 (24.7%)	0 (0%)	46 (26.4%)	4.215*	
2 – 4h	97 (52.2%)	5 (41.7%)	92 (52.9%)	0.565	
>4h	43 (23.1%)	7 (58.3%)	36 (20.7%)	8.950*	^{FE}p=0.007*
Grade of coma					
I	91 (48.9%)	0 (0%)	91 (52.3%)	12.287*	
II	67 (36%)	0 (0%)	67 (38.5%)	7.222*	
III	22 (11.8%)	6 (50%)	16 (9.2%)	17.922*	^{FE}p=0.001*
IV	6 (3.2%)	6 (50%)	0 (0%)	89.90	^{F}eb <0.001*
Duration of stay					
<2days	136 (73.1%)	9 (75%)	127 (73%)	0.023	
2-7d	46 (24.7%)	0 (0%)	46 (26.4%)	4.215*	
>one week	4 (2.2%)	3 (25%)	1 (0.6%)	31.827*	^{FE}p=0.001*
PH					
Normal	135 (72.6%)	0 (0%)	135 (77.6%)	33.955*	^{FE}p<0.001*
Decreased	51 (27.4%)	12 (100%)	39 (22.4%)		
HCO_3					
Normal	154 (82.8%)	3 (25%)	151 (86.8%)	30.081*	^{FE}p<0.001*
Decreased	32 (17.2%)	9 (75%)	23 (13.2%)		
PO_2					
Normal	148 (79.6%)	0 (0%)	148 (85.1%)	49.960*	^{FE}p<0.001*
Decreased	38 (20.4%)	12 (100%)	26 (14.9%)		
PCO_2					
Normal	146 (78.5%)	0 (0%)	146 (83.9%)	46.821*	
Decreased	35 (18.8%)	12 (100%)	23 (13.2%)	55.342*	^{FE}p<0.001*
Increased	5 (2.7%)	0 (0%)	5 (2.9%)	0.354	

χ^2: **Chi-square test** F.E.**: Fisher Exact**

TABLE (2): RELATION BETWEEN GRADE OF TOXIC COMA AND TOXIC AGENTS, MODE OF POISONING, AND TIME OF PRESENTATION.

	Grade of coma				χ^2	**p**
	I (n= 91)	**II (n= 67)**	**III (n= 22)**	**IV (n= 6)**		
Toxic agent						
Drug	73 (80.2%)	45 (67.2%)	5 (22.7%)	3 (50%)	26.413*	<0.001*
Insecticide	5 (5.5%)	9 (13.4%)	14 (63.6%)	1 (16.7%)	34.967*	<0.001*
Hashish	6 (6.6%)	2 (3%)	0 (0%)	0 (0%)	1.744	0.621
Co	0 (0%)	6 (9%)	1 (4.5%)	0 (0%)	8.893*	0.017*
Snake	5 (5.5%)	1 (1.5%)	0 (0%)	0 (0%)	2.182	0.451
Unknown	2 (2.2%)	4 (6%)	1 (4.5%)	1 (16.7%)	4.282	0.209
Dormex	0 (0%)	0 (0%)	0 (0%)	1 (16.7%)	8.675*	0.034*
Alcohol	0 (0%)	0 (0%)	1 (4.5%)	0 (0%)	6.077	0.151
Manner						
Accidental	48 (52.7%)	33 (49.3%)	7 (31.8%)	3 (50%)	3.158	0.378
Suicidal	43 (47.3%)	33 (49.3%)	15 (68.2%)	3 (50%)	3.221	0.378
Homicidal	0 (0%)	1 (1.5%)	0 (0%)	0 (0%)	3.849	0.509
Time of presentation						
Within 2h	29 (31.9%)	14 (20.9%)	3 (13.6%)	0 (0%)	5.665	0.117
2 – 4h	50 (54.9%)	30 (44.8%)	14 (63.6%)	3 (50%)	2.965	0.406
>4h	12 (13.2%)	23 (34.3%)	5 (22.7%)	3 (50%)	12.352*	0.005*

TABLE (3): RELATION BETWEEN TAC WITH GRADES OF TOXIC COMA AND CAUSATIVE TOXIC AGENT.

	N	TAC		**Test of Sig.**	**p**
		Mean ± S.D.	Median (Min. – Max.)		
Grade of coma					
I	91	0.59 ±0.13	0.56 (0.06 –0.88)	H= 56.432*	<0.001*
II	67	0.58 ±0.10	0.56 (0.25 –0.77)		
III	22	0.33 ±0.13	0.31 (0.17 –0.60)		
IV	6	0.14 ±0.15	0.10 (0.02 –0.44)		
Causative Toxic agent					
Drug	126	0.56 ± 0.14	0.56 (0.06 – 0.87)	H=9.755	0.082
Insecticide	29	0.47 ± 0.19	0.51 (0.08 – 0.72)		
Hashish	8	0.56 ± 0.11	0.56 (0.33 – 0.66)		
Co	7	0.56 ± 0.17	0.55 (0.23 – 0.77)		
Snake	6	0.62 ± 0.14	0.62 (0.45 – 0.77)		
Unknown	8	0.43 ± 0.27	0.45 (0.02 – 0.88)		
Dormex	1	0.44#			
Alcohol	1	0.19#			

U: Mann Whitney test H: H for **Kruskal Wallis test**

TABLE (4): UNIVARIATE AND MULTIVARIATE LOGISTIC REGRESSION ANALYSIS FOR THE PARAMETERS AFFECTING TOXIC COMA OUTCOME (N = 12 VS. 174)

	Univariate		#Multivariate	
	p	**OR (95%C.I)**	**p**	**OR (95%C.I)**
Age				
<15	**0.177**	0.344 (0.073 –1.618)		
15 – 40	**0.552**	1.433 (0.438 –4.688)		
40 – 60	**0.824**	0.788 (0.096 –6.463)		
>60	**0.049***	5.600 (1.000 –31.361)	0.265	0.017 (0.000 –22.445)
Male	**0.027***	10.267 (1.297 –81.248)	0.113	33.295(0.437 –2537.16)
Female	**0.027***	0.097(0.012 – 0.771)		
Toxic agent				
Drug	**0.056**	0.313 (0.095 –1.031)		
Insecticide	**0.360**	1.897 (0.481 –7.478)		
Hashish	**0.999**	–		
Co	**0.999**	–		
Snake	**0.999**	–		
Unknown	**0.049***	5.600 (1.000 –31.361)	0.195	0.066 (0.001 –4.013)
Dormex	**1.000**	–		
Hashish& alcohol	**1.000**	–		
Manner				
Accidental	**0.272**	0.500 (0.145 –1.722)		
Suicidal	**0.256**	2.047 (0.594 –7.047)		
Homicidal		–		
Time of presentation				
Within 2h	**0.997**	–		
2 – 4h	**0.455**	0.637 (0.195 –2.084)		
<4h	**0.006***	5.367 (1.609 –17.904)	0.225	4.656 (0.389 –55.746)
Grade of coma				
I	**0.996**	–		
II	**0.997**	–		
III	**<0.001***	9.875 (2.849 –34.225)	0.663	1.643 (0.176 –15.377)
IV	**0.999**	–		
Duration of stay				
<2 days	**0.879**	1.110 (0.288 –4.278)		
2 – 7 days	**0.997**	–		
>one week	**0.001***	57.667 (5.443 –610.92)	0.012*	480.438 (3.848 –59991.3)
P.H. (Acidosis)	**0.995**	–		
HCO_3	**<0.001***	19.696 (4.963 –78.157)	0.046*	24.645 (1.056 –575.003)
PO_2	**0.995**	–		
PCO_2 Normal	**0.995**	–		
PCO_2 Decreased	**0.995**	–		
PCO_2 Increased	**0.999**	–		
TAC$	**<0.001***	0.303 (0.186 –0.496)	0.004*	0.175 (0.054 –0.572)

OR: Odd's ratio C.I.: Confidence interval L.L.: Lower limit U.L.: Upper Limit
#: All variables with p<0.05 included in the multivariate *: Statistically significant at $p \leq 0.05$, $: for each 0.1 TAC.

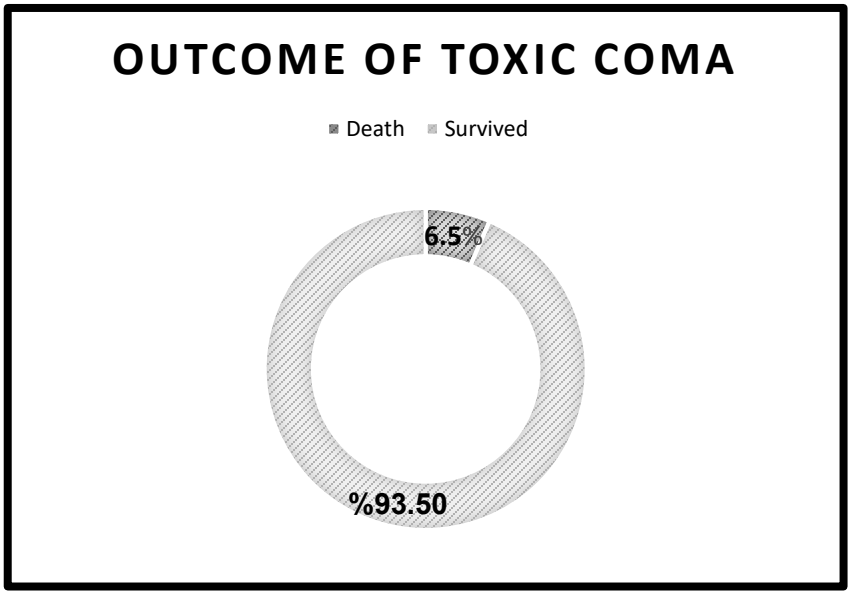

FIGURE (1): outcome of toxic coma patients

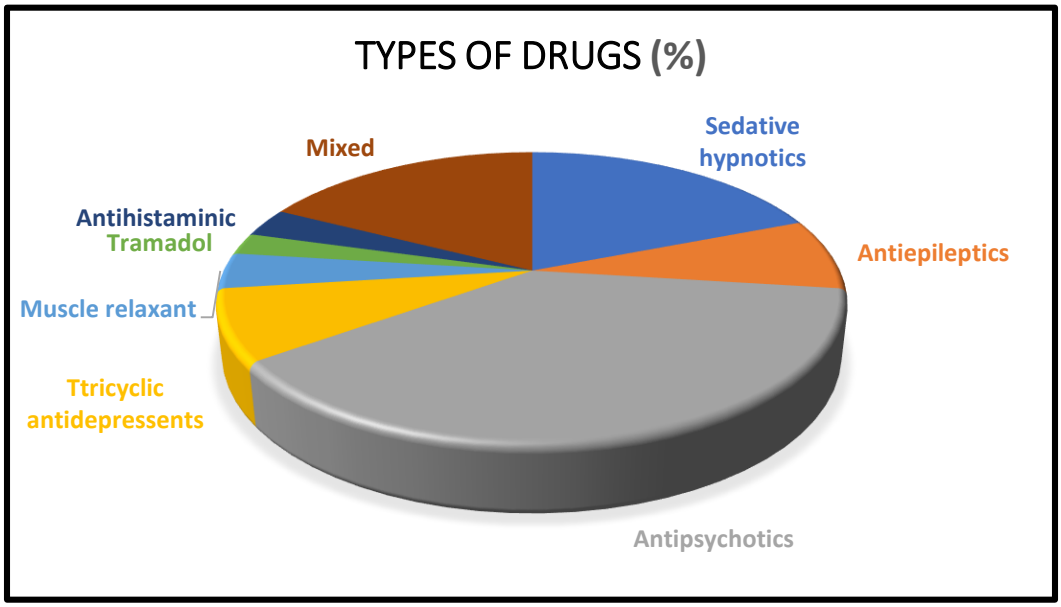

Figure (2): Types of causative toxic drugs.

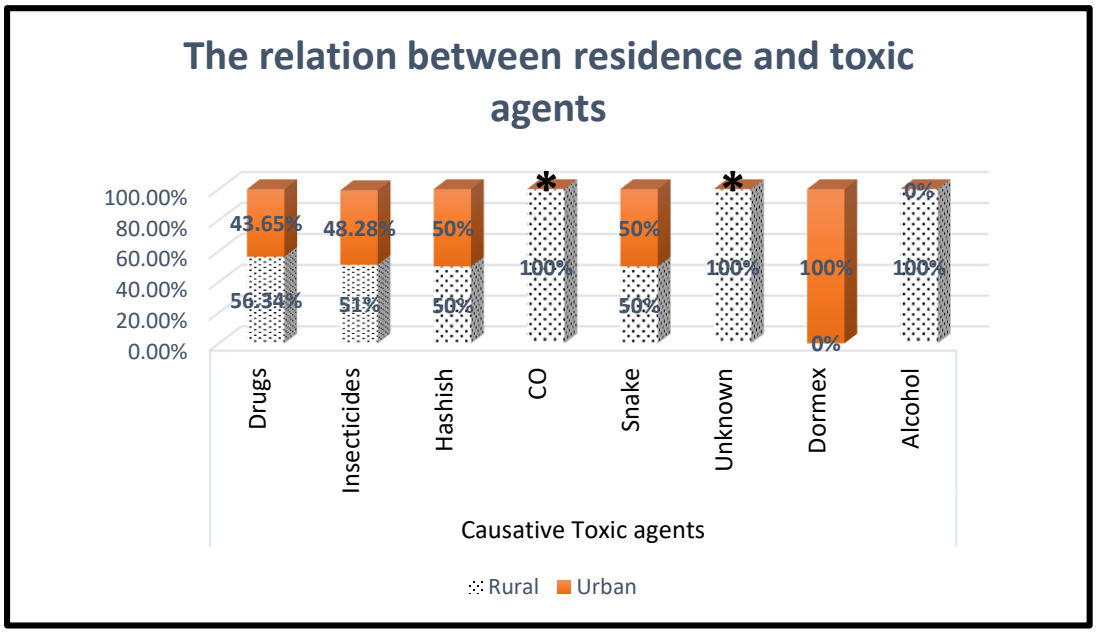

Figure (3): Relation between Residence and causative toxic agents.

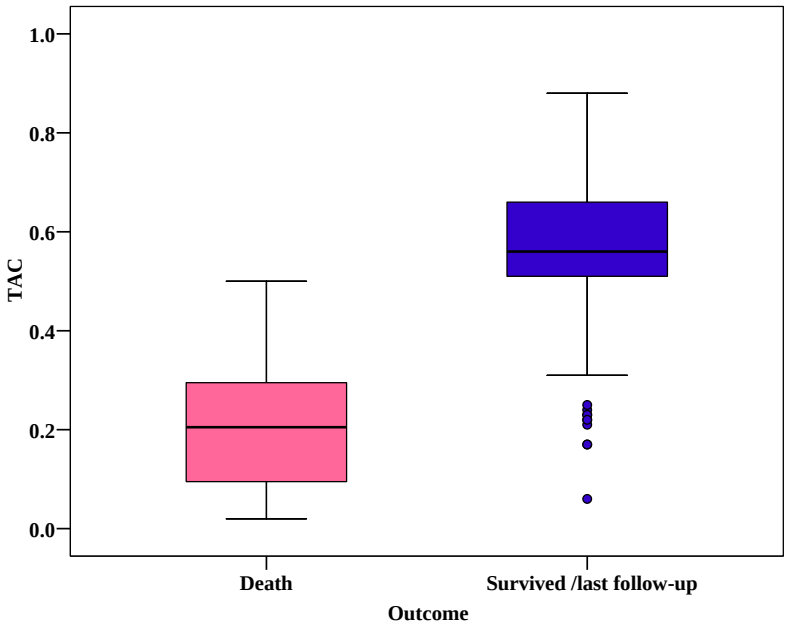

Figure (4): Relation between TAC with the outcome
(Median of group A: 0.06-0.88 & Median of group B: 0.02-0.50)

DISCUSSION

Toxic coma is a life-threatening condition, representing a challenge for toxicologists. The present study reflected the prevalence of toxic coma among the patients who arrived at Menoufia Poison Control Center (MPCC) at Menoufia University Hospital over one year (Mar 1, 2020, to the end of February 2021).

In the present study, the total number of toxic coma patients was 186; this number represented 16.42% % of all acute poisoning cases arrived at MPCC during the same study period. The mortality rate was 6.4%.

In the study of Mohamed & Gawesh in 2019, the mortality rate was higher (12%) among toxic coma patients. However, Forsberg et al. in 2009 revealed that the mortality rate was 2.8% due to toxic coma. While in the study of Moawad et al., 2015 the mortality rate was 14.2%. This disparity in mortality rates could be related to the difference in the causative toxic agents and hospital arrival time.

Because this is the age of activity, stress, and worries, 50 percent of patients in the current study were between the ages of 15 and 40; it is the age of committing suicide. There was non-significant relation between the age of toxic coma patients and the mortality in the current study; this disagrees with Mohamed & Gawesh in 2019, as they found a significant relationship between patient age and mortality. Azadi-Mood et al., 2011 also reported that increasing age was associated with fatal outcomes in a drug-induced coma due to the age groups in the prior studies being different.

The male gender predominates (54.3%) in the present study; this could be due to a higher rate of drug exposure (chronic diseases or drug addiction) and maybe exposure to more economic and social stresses than females. This finding is correlated with that of Mohammed & Gawesh 2019.

Drug overdose is the most common causative toxic agent in the current study; this is possibly due to the widespread use of drugs as it presents in nearly all homes. It is available without prescriptions, so it is easily accessible. Dadpour et al., 2017, also reported that drugs were the most frequent cause of poisoning.

However, the most common toxic agent-inducing coma in Mohammed & Gawesh in the 2019 study was organophosphorus poisoning. At the same time, opioids were the most frequent cause in the study by Mousavi et al., 2015. On the contrary, Forsberg et al., 2009 determined that alcohol is the most frequent causative agent. These differences between results in studies may be due to different study populations, different areas, and times of these studies.

Suicidal manner represented about half of toxic coma cases (50.5%); this reflects stress and troubles in daily life. It is also associated with high mortality (66.4%) and causes about 50% of coma grade IV. The person who decides to commit suicide takes a large amount of poison. Suicidal manner of poisoning is also the commonest in the research conducted by Sarivastave et al., 2005; Hassanian-Moghaddam et al., 2007; and Kann et al., 2014.

The current investigation discovered a relationship between the patient's coma grade and their mortality. All mortalities were among patients with coma grades III & IV (50% each), as a higher coma grade has associated with deteriorated system functions. This result agreed with Hassanian-Moghaddam et al., 2007 who reported that mortality was higher in grades III and IV (11% and 34%, respectively). David Bates had declared in his study that the grade of coma is predictive of mortality; the same was reported by Karcioglu et al., 2000 and Unverir et al., 2006.

Regarding the arterial blood gases (ABG) results in the present study, it revealed that acidosis, hypoxia, low HCO_3, and low PCO_2 were significantly associated with toxic coma patient's mortality, as these findings are usually associated with deteriorated hemostasis. These findings correlated with Hua et al., 2017, who claimed that acidosis was associated with mortality, although he found a high $PaCO_2$ level.

Regarding the residents in the current study, 58.6% of patients live in rural areas. There is a significant relationship between living in rural

areas and CO. toxicity and unknown poison toxicity. CO. poisoning could be due to relying on wood-fired heating at home in winter, especially in poorly ventilated spaces, while unknown agent toxicity may be due to denying the name of the toxic agent due to social considerations and sometimes legal responsibility. Marcelle et al. revealed in 2016 that most of the C.O. intoxicated cases (83.7%) were from rural areas.

In the present study, the total antioxidant capacity (TAC) was significantly low with patient mortality and coma grade IV, as toxicity usually induces oxidative stress releasing ROS and compromising antioxidant protection mechanisms and inhibiting antioxidants enzymes.

Ranjbar et al. also observed in their study in 2005 a significant correlation between Organophosphorus toxicity and reduced total antioxidant capacity.

Emam et al., in 2021**,** reported in their study on Aluminum phosphide poisoning (ALP), that the TAC was significantly low in patients died due to aluminum phosphide poisoning as organophosphorus and ALP induce oxidative stress releasing ROS and compromising antioxidant protection mechanism and inhibit of antioxidants enzymes.

CONCLUSION

The study concluded that the incidence of toxic coma was higher in the age group 15-<40 years, male gender, delayed hospital arrival, longer duration of hospitalization, residence in a rural area, suicidal manner. Drug overdose was the most common causative toxic agent, followed by insecticides. Patients living in rural areas are more susceptible to C.O. and unknown toxin poisoning.

Mortality is mainly associated with male sex, delayed hospital arrival (>4 hours), higher coma grades (III& IV), long-duration hospital stay (> one week), abnormal ABG (acidosis, low PO_2, decreased HCO_3, low PCO_2).

Factors that predispose the patients to poor outcomes and predict mortality are extended hospital stay, decreased HCO_3, delayed hospital arrival, low level of TAC.

RECOMMENDATIONS

Future studies on large-scale populations are needed to confirm the results of the present study.

TAC can be used as a prognostic marker in cases of toxic coma.

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CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest in this research**.**

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الملخص العربي

خصائص الغيبوبة التسممية ودور القدرة الكلية لمضادات الاكسدة كعلامة تنبؤية

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المقدمة: الغيبوبة هي حالة من فقدان الوعي بسبب خلل في نظام المخ. وهي تعتبر حالة تهدد الحياة وتحتاج إلى تدخل طبي فوري. القدرة الكلية المضادة للأكسدة هي علامة بيولوجية تمثل كمية جميع مضادات الأكسدة الذاتية في الدم وسوائل الجسم.

.الهدف من هذه الدراسة: تهدف هذه الدراسة إلى تحليل خصائص الغيبوبة التسممية (الناتجه عن التسمم الحاد) وتقييم الدور التنبئي لتحليل القدره الكليه الاجماليه للاكسده والمتغيرات الأخرى على مخرجات الغيبوبة التسممية.

المواد والطرق: اجريت هذه الدراسة المستقبلية على مرضي الغيبوبة الناتجه عن التسمم الحاد والذين تم حجزهم في مركز علاج التسمم فى مستشفيات جامعه المنوفيه.

النتائج: خلصت الدراسة إلى أن عدد حالات الغيبوبة التسممية خلال مدة الدراسة 186حالة. وقد كانت نسبة حدوث الغيبوبة السامة كانت أعلى في الفئة العمرية 15-40 سنة ، وفي الذكور ، وفى الحالات التي تأخرت فى الوصول إلى المستشفى (أكثر من 4 ساعات) ، والجرعة الزائدة من الأدوية كانت هي الماده السامه الأكثر شيوعًا ، يليها المبيدات الحشرية. والنمط الانتحاري قد تسبب في أكثر من نصف الحالات (50,5%)، ومدة البقاء فى المستشفي الأطول تزيد من حدوثها.

العوامل التي تتنبأ بنتائج سيئة و حدوث الوفاه ترتبط بجنس الذكور ، وتأخر الوصول إلى المستشفى (> 4 ساعات) ، ودرجات الغيبوبة الأعلى و طول فترة الاقامة في المستشفى (اكثر من أسبوع)، وتأخر الوصول وانخفاض القدرة الكلية لمضادات الاكسدة كعلامه تنبؤيه في حالات الغيبوبه التسممية.

التوصيات: نوصى بعمل دراسات أخرى متعددة على نفس الموضوع لتأكيد تلك النتائج مع امكانية استخدام القدره الكليه لمضادات الاكسده كعلامه تنبؤيه في حالات الغيبوبه التسممية.