ACUTE METHANOL POISENING: PROGNOSTIC FACTORS AND ROLE OF GLASGOW COMA SCALE

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ABSTRACT

Introduction: acute methanol poisoning is one of the most important poisonings among people with low socio-economic classes who may consume illegal alcoholic beverages as it is cheap and available causing high morbidity and mortality especially in case of delayed diagnosis or treatment. **This study aimed** to characterize the risk factors related to mortality in patients presenting with suspected methanol poisoning and to detect the efficacy of using the GCS scoring system for prediction of mortality in these patients and identifying critically poisoned patients of high risk that need rapid and aggressive treatment.

Subjects and Methods: a prospective observational study on 51 patients with acute methanol poisoning was reported at Ain-Shams poison control center-Egypt from April 2015 to April 2017. Patients were divided according to their outcome into two groups: livings and dead.

Results: Hypotension, acute respiratory failure, pulmonary edema and CGS score were significantly associated with mortality. When assessing the risk factors significantly associated with mortality according to their priority; pH $\leq$ 6.79 was the most important parameter followed by GCS score $\leq$7 and pCO2 $\geq$31.88 mmHg. There was a negative correlation between pCO2 and pH in dead. The observed mortality was not significantly different from the predicted mortality determined by GCS scoring system.

Conclusion: Hypotension, acute respiratory failure, pulmonary edema and CGS are robust markers of mortality along with pH $\leq$6.79, GCS score $\leq$7 and pCO2 $\geq$31.88 mmHg. GCS scoring system could be predictive for mortality in high risk patients.

Key words: methanol, GCS, risk factors, mortality.

INTRODUCTION

wood alcohol (Methyl alcohol) is a colorless, odorless, highly toxic organic solvent (**Pohanka., 2016**) which is a component in many industrial solvents, photocopying fluids, washing fluids, paints removers, antifreeze formulations and perfumes (**Shah et. al., 2012**). Since methanol is available and cheap, it is used in the production of illegal alcoholic beverages in many developing countries (**Giovanetti, 2013**) causing high morbidity and mortality in many methanol poisoned patients (**Kute et. al., 2012**).

Acute methanol poisoning usually occurs from intentional overdose or accidental ingestion (**Pohanka, 2016**). Methanol does not cause toxicity by itself and its pathological effects are caused by the accumulation of its metabolites formic acid which affects central nervous system, gastrointestinal tract and eyes causing metabolic acidosis and cellular dysfunction (**Sharma et. al., 2012**). Glasgow coma

scale (GCS) has been introduced as a scoring system since 1974 and it became widely used for prediction of the outcome of critically ill patients (**Davies et. al. 2008 & Grieb et. al., 2013**).

AIM OF THE WORK

This study aimed to characterize the risk factors related to mortality in patients with suspected methanol poisoning and to detect the efficacy of GCS scoring system to identify critically poisoned patients of high risk that need rapid and aggressive treatment.

SUBJECT AND METHODS

This prospective designed study was carried out in Ain Shams Poison Control Center, Ain Shams University during the period from April 2015 up to April 2017.

1. Subjects and groups:

The demographic data for all studied patients as; age, sex, being habitually alcoholic, medications and medical history as well as information related to methanol poisoning as mode (intentional or unintentional) route and time of poisoning, delay since poisoning and co-ingestions (ethanol, drugs of abuse).

Subjects were chosen according to the following criteria:

Inclusion criteria:

1- Patients diagnosed as acute methanol poisoning according to history, physical or laboratory findings.

2- Positive serum-methanol level at the time of admission (samples was taken at time of admission).

Exclusion criteria:

- Patients with negative blood methanol levels.

- History of co-ingestion of ethanol or drugs of abuse that may affect consciousness.

- Patients who were managed prior admission with buffer, antidote or mechanical ventilation that could interfere with the analysis.

Awake Patients or relatives of the patients who were unconscious were informed about the study aim and the research design. An informed consent was obtained from those who agree to participate. Reassurance of confidentially was confirmed, all data were documented by a code rather than patients name. The patients were divided into two groups according to their outcome: group I, patients who survived with or without complications; group II, dead patients.

2. Methods:

On admission (prior any treatment), the GCS score was recorded for all participated patients, blood samples were withdrawn to test the following: blood-methanol level, arterial pH, partial pressure of CO2 (pCO2) , base deficit, bicarbonate, Osmolal gap, Anion gap and serum-creatinine.

- Blood methanol level: 10 mL of heparinized venous blood sample were withdrawn from each patient and kept refrigerated at 2-4°C and processed within 6 hours after collection with Gas Chromatography (GC) according to **Wasfi et. al., (2004) & Seda et. al., (2010)**.

- Glasgow Coma Scale score: The GCS score consists of three tests: ocular, verbal, and motor responses. The lowest possible total GCS score is 3 (deep coma or death), while the highest is 15 (fully awake person) (**Teasdale and Jennett, 1974**).

- Treatment: All patients were managed under directions of the center's physicians as follow:

Correction of acidosis, Folic acid, ethanol as an antidote and hemodialysis according to standards protocol.

4- Statistical analysis: The collected data were computerized and statistically analyzed using SPSS for Windows statistical package, version 16. Quantitative data was expressed as mean and standard deviation (SD). Pearson Chi- Square test was used to compare between groups as regard categorical variables; while Mann-Whitney U-test was used to compare continuous data. Student t test was used to compare quantitative variables between 2 groups. Pearsons and Spearman Correlation Coefficient was used to find correlation between quantitative variables. In Cox regression analysis death was the only dependent variable. Receiver operating characteristic (ROC) curve, area under the curve (AUC), Standardized mortality ratio (SMR) and 95% CI were calculated. Results considered to be significant when p value ≤ 0.05.

RESULTS

1. Patient characteristics and outcome: 51 male, middle aged patients were admitted to emergency department with acute methanol poisoning after a mean latent period of 13.91 ± 5.42 hours. The baseline characteristics of the patients at time of admission, treatment and outcome is shown in table (1).

Table (1): Baseline characteristics, treatment and outcome of patients with acute methanol poisoning:

Variable	Patients (n=51)
Patient characteristics	
Age, years*	43.76 ±12.92 (22.0- 58.0)
Sex	Male (100%)
Route of methanol poisoning	Ingestion (100%)
Mode of methanol poisoning	Intentional (100%)
Alcohol consumption habit n (%)	Habitual alcohol consumption, 47 (92.2%)
Delay time, hours*	13.91 ± 5.42 (7-24)
Type of methanol poisoning	Acute poisoning (100%)
Clinical findings	
GCS score*	9.05 ± 2.15 (4 - 13)
Hypothermia, n (%)	10(19.6%)
Hypotension, n (%)	9(17.6%)
Acute respiratory failure, n (%)	9(17.6%)
Arrhythmia, n (%)	8(15.7%)
Pulmonary edema, n (%)	5(9.8%)
Laboratory findings	
Methanol level, mg/dL*	92.53±37.85(12-150)
pH*	6.97±0.85(6.34-7.4)
pCO2, mmHg*	27.15±15.38(12-75.29)
Bicarbonate mmol/L*	9.29±3(4 to 17)
Base deficit, mmol/L*	16.3±10.31(0 to 31)
Osmolal gap, mOsm/kg H_2O*	30.68±4.91(20.10-36.60)
Anion gap, mmol/L*	34.64±7.78(28.70-50.36)

Urea nitrogen, mg/dL*	24.70±4.30(19-31)
Serum creatinine, mg/dL*	1.71±0.22(1.30-2.09)
Treatment modalities	
GL n (%)	0 (0%)
Ethanol n (%)	46(90.01%)
Folic acid n (%)	51(100%)
Hemodialysis n (%)	38(74.5%)
Mechanical ventilation, n (%)	12(23.5%)
Outcome	
livings n (%)	30(58.8%)
livings with sequelae n (%)	12(23.5%)
Blindness	5(9.8%)
Diminished vision	3(5.9%)
Neurological complications	4(7.8%)
Dead n (%)	9(17.6%)

*Values are expressed as mean ± standard deviation (range).
GCS: Glasgow Coma Scale. pCO2: partial pressure of CO2.CPR:Cardiopulmonary resuscitation. GL: gastric lavage. n=number of patients. %= percent to total .

2. Analysis of the prognostic factors:

Comparison between livings and dead as regard clinical findings revealed statistical significant differences as regard hypotension, acute respiratory failure, pulmonary edema and GCS score and non-significant differences as regard age, latent period, hypothermia and arrhythmia (table 2).

Table (2): Comparison between both livings and dead as regard clinical findings using t test, Mann-Whitney U-test & Pearson Chi- Square test:

Clinical findings	Livings	Dead	test	*P* value
Age# (t=51)	41.0±14.03	47.71±10.9	t= 1.06	0.30º
Latent period# (t=51)	12.75±5.25	15.57±5.62	MW=1.13	0.27º
Hypothermia (t=10) n (%)	5 (50%)	5 (50%)	$\chi2$ =0.78	0.38º
Hypotension (t=9) n (%)	3 (33.3%)	6 (66.7%)	$\chi2$=5.13	0.02*
Arrhythmia (t=8) n (%)	5 (62.5%)	3 (37.5%)	$\chi2$=0.08	0.77º
Acute respiratory failure (t=9) n (%)	3 (33.3%)	6(66.7%)	$\chi2$=5.13	0.02*
Pulmonary odema (t=5) n (%)	0 (0 %)	5 (100%)	$\chi2$ =5.2	0.02*
GCS score# (t=51)	9.71±1.68	6.0±1.22	t=6.24	<0.001**

#= Values expressed as mean ± standard deviation n=number of cases. %= percent of number of cases to total number of cases in each parameter. t: total number of cases $\chi2$= Pearson Chi- Square test. MW =Mann-Whitney U test. GCS= Glasgow Coma Scale. t= student t test. ** highly significant. *significant. º= not significant.

As regard laboratory findings, there were statistical significant differences as regard mean values of blood methanol level, pH, pCO2, bicarbonate, base deficit, urea nitrogen and serum creatinine whereas, no statistical significant differences was found as regard mean value of osmolal gap and anion gap between livings and dead (table 3).

Table (3): Comparison between both livings and dead as regard laboratory findings using t test & Mann-Whitney U-test:

Lab. findings	Livings	Dead	test	*P* value
Methanol level,mg/dl	80.10±24.29	110.29±48.05	t= 1.98	0.04*
pH	7.19±0.2	6.58±0.16	t= 8.77	<0.001**
***p*CO2, mmHg**	23.66±11.74	43.42±18.23	MW = 2.92	0.004**
Bicarbonate, mmol/L	10.90±2.96	7.00±3.00	MW=2.41	0.01*
Base deficit, mmol/L	14.04±9.93	26.8±2.47	MW=2.01	0.02*
Osmolal gap,mOsm/kg H2O	29.52±4.15	32.34±5.74	t= 1.18	0.25°
Anion gap,mmol/L	33.53±4.03	36.50±8.29	t= 1. 32	0.21 °
Serum creatinine mg/dL	1.61±0.17	1.84±0.21	t= 2.47	0.02*
Urea nitrogen mg/dL	22.8±3.58	27.41±3.94	t= 2.51	0.02*

GCS= Glasgow Coma Scale. pCO2= partial pressure of CO2. t= student t test. MW=Mann-Whitney U test. ** highly significant. *significant. °= not significant.

Cox regression analysis revealed that pH, GCS score and pCO2 were found to be the significant prognostic factors for methanol mortality in the examined subjects. Although base deficit and bicarbonate (reflected the metabolic components of the acidosis) were also associated with mortality but both differences were dependent on pH as shown in table (4).

Table (4): Factors associated with mortality using Cox regression analysis:

Variable	OR(95% CI)	*P*-value
GCS score	0.805 (0.651-0.962)	0.028*
Methanol level	2.5 (0.32 – 19.53)	0.38 °
PH	4.765 (1.22-17.40)	0.016*
***P*CO2**	0.30 (0.12 – 0.77)	0.004*
Bicarbonate	6.75 (1.02 – 44.71)	0.01*
Base deficit	6.89 (1.22 – 44.94)	0.01*
Urea nitrogen	0.31 (0.11 – 0.63)	0.09 °
Serum creatinine	3.11 (0.41 – 23.39)	0.26 °

OR: odds ratio. 95% CI: confidence interval. °= not significant. *significant.

The ROC curve and the cut off values with the highest sensitivity and specificity for pH, GCS and pCO2 were shown in figures (1&2). pH and GCS score showed higher sensitivities and specificities than pCO2 as a predictive variable for mortality
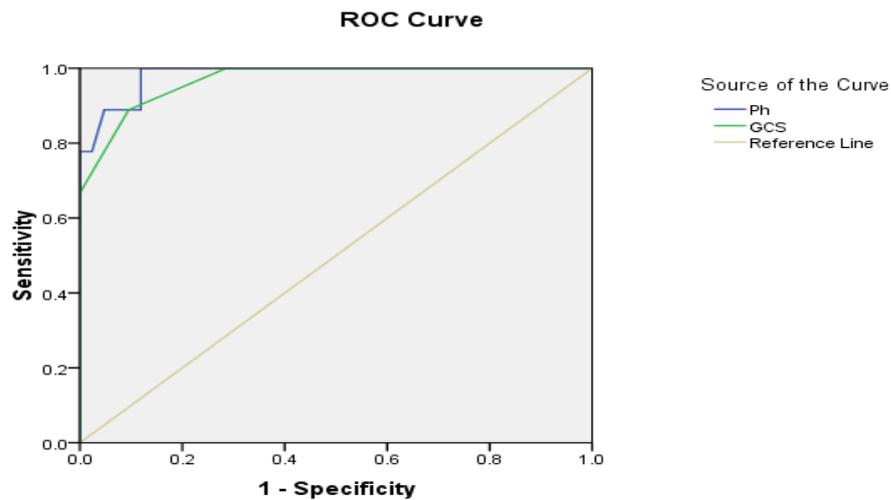
Figure (1): ROC plot showing the ability of pH and GCS scoring system to predict mortality. pH at level of 6.79 or lower can predict mortality with 0.889 sensitivity and 0.952 specificity. GCS score at 7 or lower can predict mortality with 0.889 sensitivity and 0.905 specificity in the studied acute methanol poisoned patients.
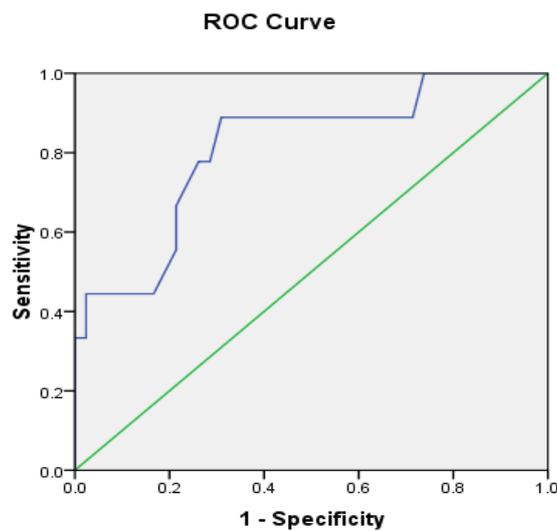

Figure (2): ROC plot showing the ability of pCO2 to predict mortality at level of 31.88 mmHg or higher with 0.778 sensitivity and 0.738 specificity in the studied acute methanol poisoned patients.

The AUC for pH, GCS and $pCO2$ with the cut off value for each variable were shown in table (5).

Table (5): The AUC for pH, GCS and pCO2 with the cut off value for each variable:

Variable	Cut off value	AUC± Std. Errorr[a]	P-value	95% CI
pH	6.79	0.983 ±0.016	<0.001**	0.952- 1.014
GCS score	7.5	0.968 ±0.025	<0.001**	0.919- 1.017
PCO2 mmHg	31.88	0.812 ±0.081	0.004**	0.653- 0.971

GCS: Glasgow Coma Scale. Highly significant. 95% CI: confidence interval. GCS=
AUC: Area under the curve**

A non-significant correlation was found between pH versus $pCO2$ values in dead ($p > 0.05$) indicating decreased ability to compensate for metabolic acidosis by hyperventilation (fig.3).
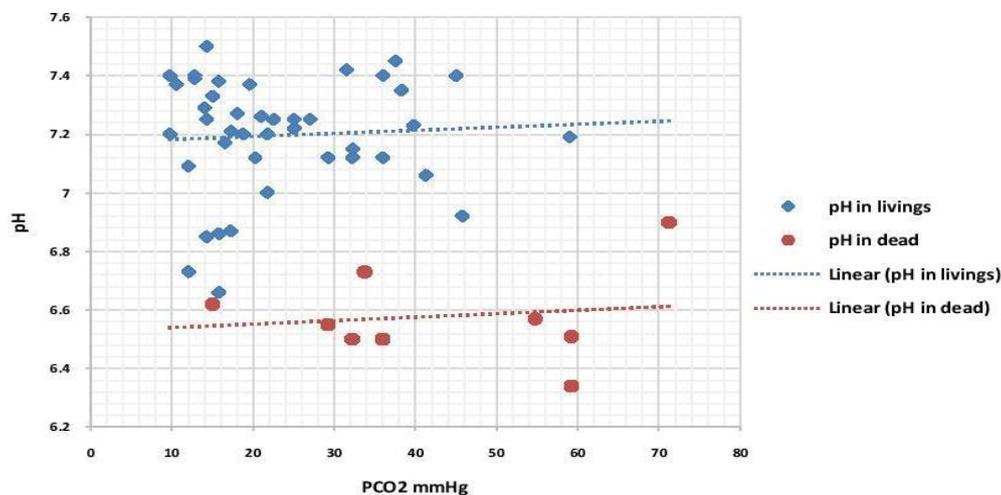

Figure (3): Scatter diagram showing correlation between pH and $pCO2$ in both livings (r= -0.040, p=0.80) and dead (r=-0.025, p= 0.95).

When the expected mortality by GCS scoring system (15.7%) and the observed mortality in the studied patients (17.6%), there was a non-significant difference between them with standardized mortality ratio (SMR) of 1.13 and 95% CI of 0.39 - 1.87 and p >0.05.

DISCUSSION

Methanol toxicity is considered a problem in many parts of the developing countries especially between people in low socioeconomic levels (**Ghanem et. al., 2015**). Methanol poisoning in Egypt still have a poor outcome because of the late arrival and diagnosis of the patients in addition to the unavailability of blood methanol level in many hospitals and the high price of fomepizole (**Rezk & Allam, 2009**). Diagnosis is based on history, signs, symptoms and the following laboratory findings: metabolic acidosis, elevated anion gap and/or osmolal gap (**Sharma et al., 2012**).

The current study was carried on 51 middle aged male patients chosen according to specific inclusion criteria. Regarding the clinical findings; hypotension, acute respiratory failure, pulmonary edema and low GCS score on admission were associated with high mortality.

As regard laboratory findings, dead had a statistically significant higher blood methanol level and lower pH than livings. *PCO2* as well as serum creatinine and urea nitrogen were found to be highly significantly increased in dead.

A wide discrepancy in blood methanol levels were noticed in many previous studies; **Shadnia et. al., (2013)** who reported a highly statistically significant methanol levels in dead when compared to survivors. On the other hand, **Hovda et. al., (2005)** found that although blood methanol levels were higher in dead than in livings, in comparison a non-significant differences were reported. **(Paasma et. al., 2007)** reported the higher means of serum methanol levels in the survivors with complications followed by the dead while the survived cases with no complications were the least. This discrepancy in blood methanol levels could be attributed to the followings; first; the retrospective design of the previously mentioned studies with its limitation. Second; the variations in treatment modalities. Third; combined ethanol and methanol which were taken by patients in these studies. That differed from the current study in being prospective study, all the cases were evaluated at admission prior to any treatment and all patients included in this study took only methanol.

pH, GCS score and *p*CO2 were significantly associated with mortality by Cox-regression with high sensitivities and specificities by ROC curve and AUC. When ranking the risk factors, pH ≤6.79 was the most important parameter followed by GCS score ≤7 then *p*CO2 ≥31.88 mmHg.

These results were consistent with **Coulter et. al., (2011)** & **Kute et. al.,** **(2012)** who stated that mortality was increased with increasing severity of metabolic acidosis. Paasma et al. (2012) concluded the strongest predictors of poor outcome after methanol poisoning to be; low pH (pH <7), coma (GCS score <8) and inadequate hyperventilation (*p*CO2 >3.1 kilopascal in spite of a pH <7) on admission. Furthermore, **Lee et. al., (2014)** found that GCS score was one of the strongest risk factors associated with mortality. **Zakharov et. al., (2017)** stated that the probability of bad outcome (death or complications) decreased with decreasing severity of metabolic acidosis. **Shadnia et. al., (2013)** stated that coma, respiratory depression, *P*CO2 were strong predictors of poor outcome but, on the other hand they reported a non-significant difference in blood pH between survivor and non-survivor groups and stated that blood H+ concentration is regulated by *P*CO2 and bicarbonate, so it depended on the compensatory situations of each patient, this study is retrospective and the number of patients is relatively small, which could be considered as a limitation of this study.

Moreover, in a study by **Rezk & Allam (2009)** entailed methanol poisoned patients admitted to the poison control centre, Ain Shams University, Egypt in one year and recorded that, mortality was much more prevalent among those who suffered from metabolic acidosis.

The importance of lack of respiratory compensation with respect to the outcome of patients was first reported by **Hovda et. al.,(2005), Paasma et. al., (2007)** and later by **Hassanian-Moghaddam et. al., (2007)**.

In this study the dead patients have lower pH values with higher levels of $pCO2$ and a negative correlation was found between pH and $pCO2$ which indicated lack of respiratory compensation that may be attributed to acute respiratory failure or pulmonary edema which had a higher incidence in dead patients or the CNS-depression evidenced by lower GCS score.

Hovda et. al., (2005) reported an inverse correlation between pH and $pCO2$ which can be explained by the co-ingestion of ethanol that caused more CNS depression and eventually loss of respiratory drive and more worsening of pH. The ability of acute methanol poisoning patients with metabolic acidosis to hyperventilate is alarm of avoiding normo-ventilation after they were put on a ventilator or a fatal worsening of the acidosis will occur **(Paasma et al.,2012)**.

CONCLUSION

From the result of this study we can conclude that Hypotension, acute respiratory failure and pulmonary edema were robust markers of mortality in the studied acute methanol poisoned patients along with pH $\leq$ 6.79, GCS score $\leq$7 and $pCO2 \geq$ 31.88 mmHg. GCS scoring system was found to detect those patients who were at risk so, GCS scoring system could be an easy predictive model in acute methanol poisoning. This study is a pilot study, larger scale studies over several years is recommended to validate the results.

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الملخص العربي

التسمم الحاد بالكحول الميثيلي : عوامل الخطوره ودور معيار جلاسكو

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المقدمه: يعتبر التسمم الحاد بالكحول الميثيلي من اخطر انواع التسمم خاصه بين افراد المجتمعات الفقيره والذي تتوفر به انواع من الكحوليات المخلوطه بالكحول الميثيلي وذلك لرخص ثمنها وسهوله العثور عليها مما يتسبب في حدوث اضرار صحيه قد تصل الي الوفياة بين هؤلاء الافراد. هدفت هذه الدراسة إلى تقييم وترتيب العوامل التي يتزامن وجودها مع حدوث وفيات بين المرضي المتعاطيين للكحول الميثيليوترتيبها وفقًا للأولوية وكذلك تقييم استعمال معيار جلاسكو كأحد هذه العوامل ودوره في تحديد المرضي ذوي الحالات الحرجه والتي تحتاج الي علاج سريع. تم عمل هذه الدراسة للمرضى الذين يعانون من التسمم الحاد بالميثانول المستقبلين في مركز مكافحة السموم جامعة عين شمس من أبريل 2015 إلى أبريل 2017. تم تقسيم المرضى إلى مجموعتين, المجموعة الأولى: المرضى الذين تم شفائهم والمجموعة الثانية: المتوفيين. من خلال نتائج هذه الدراسه وجد ان انخفاض ضغط الدم ,فشل التنفس الحاد وتورم الرئتين من العوامل التي ارتبطت بزياده نسبه الوفيات و كذلك وجد ان الرقم الهيدروجيني≥ 6.79 من الممكن اعتباره أقوى العوامل المؤديه الي الوفاة يليه معيار جلاسكو ≥ 7.5 وفي النهاية $pCO2 \geq 31.88 mmHg$.