Erciyes Med J 2021; 43(1): 13–9 • DOI: 10.14744/etd.2020.52993
ORIGINAL ARTICLE – OPEN ACCESS

Impaired Renal Function in Outpatients with Acute Carbon Monoxide Poisoning

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ABSTRACT

Objective: The study aims to investigate the incidence of early stage kidney disease in outpatients with acute carbon monoxide poisoning (CMP) and to assess whether the Chronic Kidney Disease Epidemiology Collaboration formula (CKD-EPI) values of these patients had any impact on the time taken for their discharge.

Materials and Methods: This retrospective case–control study consisted of a total of 94 patients (47 patients with carbon monoxide poisoning induced by exposure to incompletely burned coal, and 47 age-and sex-matched controls). Patient details including age, CKD-EPI value, hemoglobin, carboxyhemoglobin (COHb), lactate, pH, SaO_2, pO_2 pCO_2, BE, HCO_3, glucose, AST, ALT, amylase, ALP, BUN, creatinine, Na, K, Cl, CK, CK-MB, troponin, uric acid levels and duration of follow-up were recorded. The post-hoc power analysis was calculated using G*Power 3.1.9.2 based on the difference between the two means by Wilcoxon–Mann–Whitney test. A Kaplan–Meier survival curve was plotted and test of equality of survival distributions for the different levels of CKD-EPI was compared.

Results: The mean age and mean COHb values of the study group were 37.24±12.91 and 21.45±9.89, respectively. The CKD-EPI values were lower in patients belonging to the CMP group (101.39±19.53) compared to the control group (114.92±14.81) (p=0.017), 92% power level, d=0.781, 95% confidence intervals (CI) on the difference between means (-23.01/-4.04). The duration of follow-up of patients with CMP was 29.04±9.7 hours. There was a significant relationship between the follow-up duration of patients with CMP in the emergency department and their CKD-EPI levels (Log-rank/Mantel–Cox, p<0.001).

Conclusion: CKD-EPI-values are reduced as a result of impaired renal function in outpatients with CMP in the early stages. Thus, long-term nephrology follow-up should be performed in outpatients diagnosed with CMP. Further investigations may be required to rule out possible chronic kidney disease (CKD) associated with impaired renal function induced by acute CMP.

Keywords: Acute toxicity; carbon monoxide poisoning; renal function; CKD-EPI; creatinine; uric acid

Cite this article as:
Ekici Günay N, Kale A, Günay N. Impaired Renal Function in Outpatients with Acute Carbon Monoxide Poisoning. Erciyes Med J 2021; 43(1): 13-9.

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Submitted
19.04.2020

Accepted
07.07.2020

Available Online Date
01.12.2020

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INTRODUCTION

CMP is a medical emergency caused by exposure to carbon monoxide released into the environment, usually due to incomplete combustion of carbonaceous substances. There is high rate of mortality in the acute period as well as chronic sequelae in survivors (1, 2). Due to high hospital occupancy rates it is common knowledge that especially during the acute period of 24–48 hours, CMP cases are observed and discharged from the emergency department itself. Those patients who do not demonstrate clinical signs or symptoms of sufficient severity necessitating hospitalization are referred to as outpatients with CMP (3). Although obvious long-term neurological effects in patients with acute CMP are described (4), there is not enough data regarding possible long-term renal impairment in patients with acute CMP, especially in the outpatient group. Certain recent studies have highlighted the fact that acute CMP could pose a potential threat in the development of chronic renal damage (5). Additionally, there is insufficient data regarding renal function in patients with acute CMP without acute kidney injury. Published literature regarding acute CMP-related renal damage consists mostly of case reports in patients with overt acute renal failure (6). In summary, there is little or no information about renal function during the acute period of CMP, especially in the outpatient group. In other words, renal function of outpatients with acute CMP may not receive due consideration and might in fact be considered to be normal.

CKD-EPI or the 4–variable version of the Modification of Diet in Renal Disease study (MDRD) is used to determine estimated glomerular filtration rate (eGFR), which plays an important role in the evaluation of renal function. BUN and creatinine are also taken into consideration in the management of emergency patients (7). The CKD-EPI value is believed to be more precise and accurate in predicting the eGFR compared to MDRD, but MDRD has low bias (8). CKD-EPI, which is used during emergency patient management, has also been the subject of some research articles, although infrequent (9). In this context, the present study aims to evaluate renal function during the early stages of acute CMP in outpatients.

MATERIALS and METHODS

This retrospective case–control study was conducted in outpatients with acute CMP in compliance with the inclusion criteria of study at the Emergency Department of Erciyes University between January 09, 2016 and 01.03.2019 after the approval of the local ethics committee (2018/656). The diagnosis and treatment of patients in the emergency department were not affected by this effort; patient data was obtained from the hospital data system, and research data was acquired. This work has been conducted in full accordance with the ethical standards of the responsible institutional or national committees on human subjects as well as the Helsinki Declaration.

Study Design

Outpatients with acute CMP with a history of using coal for heating purposes and those with a COHb level of 5% or more were included in the study. Any underlying systemic pathology, especially cardiopulmonary, was considered to be exclusion criteria regardless of serum COHb level. The study group was selected carefully, and patients with history of alcohol intake, smoking, and/or drug use were excluded. Other exclusion criteria included the loss of data related to the evaluation of renal function, which was considered to be the cornerstone of this study, and patients who did not have records for CKD-EPI, BUN, and/or creatinine values were excluded from the study. Pregnancy, pediatric age group, patients requiring hospitalization, and death were determined to be additional exclusion criteria. Acute treatment and follow-up of most CMP cases, which we define as outpatient poisoning, are performed in emergency departments due to hospital occupancy rates. Inpatient admission to the hospital at our location for CMP requires presence of specific features such as brain edema, coma, acute coronary syndrome, acute kidney injury, hyperbaric oxygen demand and underlying comorbidities. Hospitalized patients have therefore not been included in the study.

The control group, which was of comparable age and sex with the study group, was selected from healthy volunteers who donated blood. Since arterial blood gas analysis could not be performed in healthy volunteers, standard reference values were taken into consideration. Some variables including age, Hb, COHb, lactate, pH, saO_2, pO_2 pCO_2, BE, HCO_3, glucose, AST, ALT, amylase, ALP, BUN, creatinine, CKD-EPI, Na, K, Cl, CK, CK-MB, troponin, and uric acid were recorded for both groups. The follow-up duration (hours) of patients with CMP from the time of admission until discharge was recorded. Samples taken for serum and arterial blood gas analysis of patients with CMP were the initial values obtained at the time of admission, and repeat measurements of these variables were not performed in most patients.

Laboratory Measurements and CKD-EPI Calculation

Arterial blood gas analysis was performed with Siemens RAPIDlab 1265 Systems (Germany) analyzers. Serum creatinine levels were measured on the Roche Cobas 8000 analyzer using the enzymatic method (CREA plus, Cobas 8000, Roche Diagnostic, USA). Measurements of glucose, AST, ALT, amylase, ALP, BUN, Na, K, Cl, CK, CK-MB, and uric acid were done on the same device using the original analyzer kits. Serum troponin levels were measured in high sensitivity cardiac troponin T form in a Roche Cobas 602 analyzer (Roche Diagnostic, USA). CKD-EPI calculation was based on age, sex, and serum creatinine values and was calculated using the original formula (10).

Table 1. Descriptive statistics of variables

Variables	n	Mean±SD	Min.–Max.
Age (years)	94	37.24±12.91	17.00–66.00
Follow-up time			
duration (hour)	47	29.04±9.7	12.00–48.00
COHb (%)	47	21.45±9.89	5.60–42.70
Hb (g/dL)	47	14.20±1.37	11.70–16.20
Lactate (mEq/L)	47	1.79±1.00	0.02–5.14
pH	47	7.45±0.04	7.38–7.57
saO_2 (%)	47	97.06±2.70	86.50–99.80
pO_2 (mmHg)	47	122.10±51.60	41.00–240.80
pCO_2 (mmHg)	47	30.41±5.93	16.10–41.30
BE (±)	47	-2.07±2.90	-11.70–2.40
HCO_3 (mmol/L)	47	21.61±2.84	14.60–26.90
Glucose (mg/dL)	94	107.26±33.24	11.00–277.00
AST (U/L)	94	18.96±5.87	10.00–36.00
ALT (U/L)	94	18.19±9.99	5.00–51.00
Amylase (U/L)	63	57.47±19.89	10.00–115.00
ALP (U/L)	90	68.48±19.03	43.00–108.00
BUN (mg/dL)	94	12.96±4.93	5.90–26.70
Creatinine (mg/dL)	94	0.76±0.18	0.38–1.16
CKD_EPI			
(mL/min/1.73 m^2)	94	108.15±18.47	71.99–144.39
Na (mmol/L)	94	139.65±2.66	132.00–145.00
K (mmol/L)	94	4.28±0.44	3.41–5.10
Cl (mmol/L)	94	102.33±3.34	96.00–116.00
CK (U/L)	79	147.36±188.23	46.00–1213.00
CK_MB (U/L)	79	32.36±17.12	6.20–109.00
Troponin (mg/mL)	94	0.0038±0.0029	0.001–0.014
Uric acid (mg/mL)	94	4.26±1.26	2.40–8.20

SD: Standard deviation; Min.: Minimum; Max.: Maximum; CoHb: Carboxyhemoglobin; BE: Base excess; AST: Aspartate aminotransferase; ALT: Alanine Aminotransferease; ALP: Alkaline phosphatase; BUN: Blood urea nitrogen; CKD_EPI: Chronic kidney disease-epidemiology; CK_MB: Creatine kinase myocardial band; Na: Sodium; K: Potassium; CK: Creatine kinase; Cl: Chlorine

Statistical Analysis

The data was analyzed with IBM SPSS Statistics 25.0 (IBM Corp. Released 2017. IBM SPSS Statistics for Windows, Version 25.0. Armonk, NY: IBM Corp.) statistical package program. Descriptive statistics were given as number of units (n), percentage (%), arithmetic mean±standard deviation (Mean±SD), minimum value (min), maximum value (max), median (M), 25th percentile (Q_1), and 75th percentile (Q_3). The normality distribution of the data for the numerical variables was checked with the Shapiro Wilk test and Q-Q graphs. An independent two

Table 2. Comparison of variables according to the groups

	Groups						
	Carbon monoxide poisoning			Healthy Controls			
	n	Mean±SD M(Q_1–Q_3)	Min.–Max.	n	Mean±SD M(Q_1–Q_3)	Min.–Max.	Test statistics+ p
Age (years)	47	38.04±13.92 34 (30–51)	18–66	47	36.44±12.03 35 (28–44)	17–66	t=0.450 p=0.655
Glucose (mg/dL)	47	111.96±15.15 111 (105–120)	81–150	47	102.56±44.47 95 (86–117)	11–277	U=211.500 **p=0.008**
AST (U/L)	47	20.3±5.29 20 (16.8–21)	13–34	47	17.63±6.22 17(13–19)	10–36	U=233.000 **p=0.023**
ALT (U/L)	47	18.94±10.11 16 (12.6–22.7)	7–51	47	17.44±9.99 15 (10–21)	5–50	U=325.500 p=0.499
Amylase (U/L)	29	75.44±23.92 81 (53–93.5)	45–115	47	51.48±14.41 53(42–59)	10–81	t=2.839 **p=0.018**
ALP (U/L)	43	69.57±19.46 71 (52–84)	43–108	47	67.56±18.98 65 (49–81)	43–103	t=0.369 p=0.714
Na (mmol/L)	47	140.26±2.52 141 (139–142)	134–145	47	139.04±2.7 139 (138–141)	132–144	t=1.721 p=0.091
K (mmol/L)	47	4.23±0.4 4.27 (3.89–4.58)	3.41–4.92	47	4.32±0.48 4.4(3.9–4.7)	3.5–5.1	t=–0.738 p=0.464
Cl (mmol/L)	47	101.89±2.64 102 (100–103)	97–108	47	102.78±3.93 103 (100–105)	96–116	U=301.000 p=0.269
CK (U/L)	43	186.7±238.7 125 (77–182)	47–1213	36	90.81±20.2 96.5 (73.25–102)	46–126	U=119.500 p=0.065
CK_MB (U/L)	43	36.57±20.41 30.3 (25–47)	6.2–109	36	26.31±8.07 28.5(17.5–32)	12–37	U=127.500 p=0.106
Troponin (mg/mL)	47	0.0049±0.0033 0.003 (0.003–0.005)	0.003–0.014	47	0.0028±0.0002 0.003 (0.001–0.003)	0.001–0.011	U=165.500 **p<0.001**
Uric acid (mg/dL)	47	4.76±1.4 4.8 (3.8–5.4)	2.4–8.2	47	3.76±0.88 3.7 (3.0–4.5)	2.4–5.5	t=3.135 **p=0.003**
BUN (mg/dL)	47	14.1±4.81 13.2(10.5–17.7)	6.9–26.7	47	11.83±4.87 11.3 (7.5–15)	5.9–21.9	t=1.727 p=0.090
Creatinine (mg/dL)	47	0.82±0.17 0.81 (0.66–0.95)	0.53–1.16	47	0.71±0.17 0.69 (0.57–0.82)	0.38–1.1	t=2.258 **p=0.028**
CKD_EPI (mL/min/1.73m^2)	47	101.39±19.53 94.1 (85.12–118.23)	71.99–135.42	47	114.92±14.81 114.55 (105.1–126.08)	84.5–144.39	U=226.000 **p=0.017**

*: Test statistics: +: Independent sample t test; †: Mann Whitney U Test; SD: Standard deviation; Min.: Minimum; Max.: Maximum; Q: Quartile; AST: Aspartate aminotransferase; ALT: Alanine Aminotransferease; ALP: Alkaline phosphatase; BUN: Blood urea nitrogen; CKD_EPI: Chronic kidney disease-epidemiology; CK_MB: Creatine kinase myocardial band; Na: Sodium; K: Potassium; Cl: Chlorine; CK: Creatine kinase

sample t-test was used for the variables that provided a normal distribution assumption in the comparisons between the groups, and the Mann-Whitney U test was used for the variables that did not provide a normal distribution assumption. Cumulative incidence of endpoint event was calculated with the Kaplan–Meier method, incidence curves were plotted and compared with a Tarone–Ware, Breslow (Generalized Wilcoxon) and Log-Rank tests. A Kaplan–Meier life curve was plotted and test of equality of survival distributions for the different levels of CKD-EPI was compared. The endpoint event was taken as discharge from the hospital (emergency department). The relationships between variables were investigated by Pearson and Spearman correlation analysis. A value of $p<0.05$ was accepted as statistically significant.

Table 3. Some basic correlations related to carboxyhemoglobin

	CoHb	BUN	Creatinine	CKD_EPI
COHb (%)				
r_s	1.000			
p	–			
BUN (mg/dL)				
r_s	-0.029	1.000		
p	0.886	–		
Creatinine (mg/dL)				
r_s	0.080	**0.540****	1.000	
p	0.692	<**0.001**	–	
CKD_EPI (mL/min/1.73 m^2)				
r_s	-0.271	**-0.443****	**-0.696****	1.000
p	0.171	<**0.001**	<**0.001**	–

r_s: Spearman's correlations coefficient; CoHb: Carboxyhemoglobin; BUN: Blood urea nitrogen; CKD_EPI: Chronic kidney disease-epidemiology

RESULTS

Descriptive Statistical Analysis of Variables

A total of 94 individuals consisting of 47 patients in the study group (24 female, 23 male) and 47 in the control group were analyzed. The mean and standard deviation values of age and COHb levels were 37.24±12.91 and 21.45±9.89, respectively. Mean and standard deviation values of duration of follow-up of patients with CMP at the emergency department were 29.04±9.7 hours. While the minimum duration was 12 hours, the maximum length of stay in the emergency department was 48 hours. There was a significant relationship between the follow-up duration of patients with CMP in the emergency department and CKD-EPI levels (Log-Rank/Mantel–Cox), p<0.001). Descriptive statistical information for other variables is given in Table 1. In addition, all ECGs recorded at the time of admission of patients in the study group were reported as normal. No pathological findings were noted in the radiological interpretations of 12 cases.

Comparative Results of Variables According to Groups

The mean and standard deviation values of serum glucose, AST, amylase, creatinine, troponin, and uric acid in the study group were 111.96±15.15 mg/dL, 20.3±5.29 U/L, 75.44±23 U/L, 0.82±0.17 mg/dL, 0.0049±0.0033 mg/mL, and 4.76±1.4 mg/dL, respectively. The values of these parameters/variables in the control group were 102.56±44.47 mg/dL, 17.63±6.22 U/L, 51.48±14.41 U/L, 0.71±0.17 mg/dL, 0.0028±0.0002 mg/mL, and 3.76±0.88 mg/dL, respectively. In the CMP group, these variables were found to be elevated and statistically significant compared to the control group. CKD-EPI values were also reduced in the CMP group (101.39±19.53) compared to healthy controls (114.92±14.81). No difference was observed between the groups in the analysis of other parameters (Table 2, 3). Variables such as age, glucose, AST, ALT, amylase, ALP, BUN, creatinine, CKD-EPI, Na, K, Cl, CK, CK-MB, troponin and uric acid, the effect size, strength, and 95% confidence intervals (CI) within parenthesis were analyzed. The post-hoc power analysis and effect size were calculated using G*Power 3.1.9.2 [14] based on difference between the two means by Wilcoxon–Mann–Whitney test (two group) to detect any significant differences between the groups for CKD-EPI at 92% power level, d=0.781, 95% CI of difference (–23,01/–4,04), effect size and type I error of 0.05. (Table 4).

Table 4. The effect size, strength, and 95% confidence interval values of the variables

			% Confidence interval of the difference	
	Effect size (d)	Power	Lower	Upper
Age	0.160	0.296	-5.51	8.69
Amylase (U/L)	1.260	0.995	5.16	42.77
ALP (U/L)	0.108	0.247	-8.94	12.96
Na (mmol/L)	0.500	0.721	-0.20	2.65
K (mmol/L)	0.205	0.351	-0.33	0.15
Uric acid (mg/dL)	1.059	0.983	0.35	1.63
BUN (mg/dL)	0.471	0.691	-0.37	4.92
Creatinine	0.614	0.824	0.01	0.19
Glukoz	0.282	0.452	-9.01	27.82
AST	0.543	0.764	-0.49	5.82
ALT	0.105	0.244	-3.99	6.99
Cl	0.392	0.597	-2.71	0.94
CK	0.568	0.786	-7.75	199.52
CK_MB	0.656	0.853	0.65	19.87
Troponin	0.898	0.957	0.0006	0.003
CKD_EPI	0.781	0.919	-23.01	-4.04

BUN: Blood urea nitrogen; AST: Aspartate aminotransferase; ALT: Alanine Aminotransferease; ALP: Alkaline phosphatase; CK_MB: Creatine kinase myocardial band; CKD_EPI: Chronic kidney disease-epidemiology; Na: Sodium; K: Potassium; Cl: Chlorine; CK: Creatine kinase

Correlation Analysis

There was no statistically significant relationship between COHb and BUN (p=0.886, rs: –0.029), between COHb and creatinine (p=0.692, rs: 0.080), and between COHb and CKD-EPI (p=0.171, rs: –0,271). However, there was a strong negative correlation between COHb and amylase measurements (p=0.013, rs: –0.783) and a moderate degree of positive correlation between COHb and ALP (p=0.037, rs: 0.438). A strong positive correlation was also found between creatine and uric acid (p<0.001, rs: 0.472) (Table 3 and 5). All correlation analyses are given in Table 5.

DISCUSSION

Main Study Finding

The most striking finding of the study was that serum creatinine levels with CKD-EPI, which are the most important indicators of renal function in the emergency department (11) were significantly different between the study and control groups. Moreover,

Table 5. The relationships between some variables in CMP group

	CoHb	BUN	Creatinine	CKD_EPI		CoHb	BUN	Creatinine	CKD_EPI
Age					ALT				
r_s	-0.007	0.324	0.131	-0.656	r_s	-0.292	0.200	0.309	-0.105
p	0.972	**0.017**	0.346	<**0.001**	p	0.139	0.148	**0.023**	0.449
Hb					Amylase				
r_s	-0.274	0.386	0.427	0.089	r_s	-0.783	-0.007	0.264	-0.237
p	0.167	**0.047**	**0.026**	0.660	p	**0.013**	0.966	0.119	0.164
Lactate					ALP				
r_s	0.329	-0.144	0.205	-0.258	r_s	0.438	0.030	0.075	-0.161
p	0.094	0.472	0.304	0.194	p	**0.037**	0.838	0.604	0.264
pH					Na				
r_s	0.044	-0.158	-0.112	0.256	r_s	-0.035	0.411	0.587	-0.256
p	0.826	0.432	0.577	0.197	p	0.864	<**0.001**	<**0.001**	0.062
saO_2					K				
r_s	-0.219	-0.128	-0.05	0.291	r_s	-0.005	0.056	0.236	-0.252
p	0.271	0.525	0.803	0.141	p	0.982	0.687	0.085	0.066
pO_2					Cl				
r_s	-0.169	-0.053	-0.076	0.092	r_s	0.100	-0.051	-0.051	0.028
p	0.401	0.794	0.705	0.650	p	0.619	0.715	0.712	0.838
pCO_2					CK				
r_s	-0.328	0.588	0.482	-0.258	r_s	0.022	0.356	0.314	-0.029
p	0.095	<**0.001**	**0.011**	0.194	p	0.922	0.026	0.052	0.861
BE					CK_MB				
r_s	-0.186	0.392	0.182	-0.031	r_s	0.065	0.292	0.230	-0.077
p	0.353	0.043	0.363	0.880	p	0.769	0.071	0.159	0.643
HCO_3					Troponin				
r_s	-0.223	0.298	0.095	0.079	r_s	0.143	0.283	0.426	-0.48
p	0.264	0.131	0.637	0.696	p	0.477	**0.038**	<**0.001**	<**0.001**
Glucose					Uric acid				
r_s	0.343	0.137	0.013	-0.209	r_s	-0.084	0.222	0.472	-0.242
p	0.080	0.322	0.928	0.129	p	0.677	0.106	<0.001	0.077
AST									
r_s	-0.25	0.351	0.331	-0.166					
p	0.209	**0.009**	**0.014**	0.231					

r_s: Spearman's correlations coefficient; CoHb: Carboxyhemoglobin; BUN: Blood urea nitrogen; CKD_EPI: Chronic kidney disease-epidemiology; CK_MB: Creatine kinase myocardial band; BE: Base excess; AST: Aspartate aminotransferase; ALT: Alanine Aminotransferease; ALP: Alkaline phosphatase; Hb: Hemoglobin; HCO_3: Bicarbonate; Na: Sodium; K: Potassium; Cl: Chlorine; CK: Creatine kinase

the study group consisted of outpatients with CMP who were not hospitalized. Compared to healthy controls, low CKD-EPI and elevated creatinine in patients with CMP indicate a significant decrease in eGFR in the CMP group. Although the significance of reduced renal function in patients with CMP during the acute stage is unclear, we think it is necessary to investigate impaired renal function associated with CMP, and this study might serve as a guide to nephrology research regarding similar cases in the future. When patients with CMP were examined and/or their laboratory findings were analyzed, it was observed that there was no prerenal, renal or post renal etiology for the reduced eGFR. Prerenal azotemia, which is the most common and most probable cause of reduced eGFR in emergency departments, was not seen in the individuals participating in the research. Elevated serum uric acid levels seen in patients with CMP, which is an important indicator that renal function is adversely affected in addition to low CKD-EPI and elevated creatinine in acute CMP, is also a notable finding. Uric acid, a final product in purine metabolism, may be expected to increase in serum for many reasons (12), but most of these reasons were ruled out in our cases. The presence of metabolic and lactic acidosis in CMP may result in elevated serum uric acid levels, but findings of blood gas analysis (in Table 1) did not demonstrate any significant component of acidosis in patients with CMP. Moreover, as the investigated cases were outpatient, acidosis was not a main finding. Although there was no clear evidence that CMP increases serum uric acid levels by reducing eGFR, we are unable to offer any other reason explanation for the elevation of uric acid. A strong positive association (Table 5) between creatinine and uric acid has been

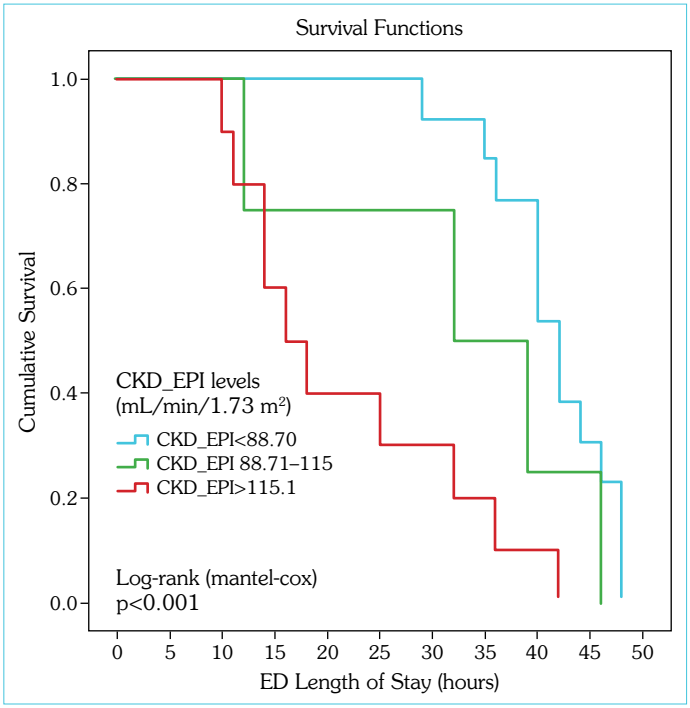

Figure 1. Kaplan-Meier survival plots for emergency department lenght of stay by CKD_EPI levels

thought to be associated with significant elevation in serum uric acid levels possibly due to reduced eGFR related to CMP. In other words, the correlation between uric acid and creatine strongly supports our hypothesis that renal function is adversely affected in acute CMP. The findings of our study therefore suggest that CKD-EPI levels in outpatients with CMP at the time of admission could serve as an important indicator of renal function which could determine the course of these patients in terms of their total follow-up time and prognosis (Fig. 1).

Secondary Study Findings

Although it is believed that the elevations in glucose, amylase, AST, and troponin (Table 2) did not affect the clinical status of patients in the acute period, the requirement for long-term follow-up of these patients was evaluated. It is possible to connect mild levels of hyperglycemia to stress. Salivary gland-induced hyperglycemia has been reported in CMP (13) and it does not appear to have any additional clinical significance in the acute period. Although elevations of AST in CMP have been reported due to prolonged loss of consciousness (14), the consciousness levels of all patients in the study were clear, and they were seen as outpatients. Therefore, it is possible that reasons other than prolonged loss of consciousness could have led to elevated AST levels in acute CMP. Troponin elevation seen in acute CMP, especially in outpatients who have no history of heart, kidney, and central nervous system disease was not evaluated as a finding of the acute event. There are reports that carbon monoxide in acute CMP causes toxicity independent of the hypoxic effect in the myocardium (15). High troponin levels seen during the clinical management of outpatient cases were not considered as an acute pathological finding in CMP. However, these findings suggest that the pathophysiological and clinical significance of troponin elevation in CMP poisoning should be further investigated, and patients with acute CMP may need to be followed-up in the long term for the development of diabetes mellitus, ischemic heart disease, liver, pancreatic and salivary gland diseases.

Limitations

Serum measurements of parameters/variables, especially CKD-EPI and creatinine, were performed only once, and baseline values could not be obtained. Creatinine does not reflect real-time dynamic changes in GFR that occur in association with acute reduction in kidney function. No other imaging or functional investigations could be performed to provide additional information regarding renal function. Although all patients in the study group appeared to be patients with acute CMP, the presence of underlying genetic disease or an organ anomaly might have affected the findings. This paper does not provide sufficient information about the short- and long-term prognosis of the acute kidney event, nor does it explain the significance of hyperamylasemia in the short and long term.

CONCLUSION

Acute CMP is associated with impaired renal function. More specifically, in patients with acute CMP, CKD-EPI is reduced due to carbon monoxide effect. Since no major causes for increased uric acid levels were identified, elevated uric acid levels observed in patients with CMP further support our hypothesis of renal impairment in acute CMP. Even in outpatients with acute CMP, renal function can be carefully and comprehensively evaluated for long-term effects. Furthermore, the risk of CKD associated with pure CMP should be the subject of future research projects. Likewise, investigations related to CMP sequelae in terms of hyperamylasemia and hepatobiliary pathologies should also be undertaken.

Acknowledgements: We would like to thank Assoc. Prof. Dr. Ferhan Elmalı for his statistical analyses.

Ethics Committee Approval: The Erciyes University Clinical Research Ethics Committee granted approval for this study (date: 19.12.2018, number: 2018/656).

Informed Consent: Written informed consent was obtained from patients who participated in this study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – NEG, AK, NG; Design – NEG, AK, NG; Supervision – NEG, AK, NG; Resource – NEG, AK, NG; Materials – NEG, AK, NG; Data Collection and/or Processing – NEG, AK, NG; Analysis and/or Interpretation – NEG, AK, NG; Literature Search – NEG, NG; Writing – NG; Critical Reviews – NEG, AK, NG.

Conflict of Interest: The authors have no conflict of interest to declare.

Financial Disclosure: The authors declared that this study has received no financial support.

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