Dicle Tıp Dergisi /
Dicle Medical Journal

2015; 42 (3): 299-304
doi: 10.5798/diclemedj.0921.2015.03.0578

ORIGINAL ARTICLE / ÖZGÜN ARAŞTIRMA

Comparison of carbon monoxide poisonings originated from coal stove and natural gas and the evaluation of Neutrophil/Lymphocyte ratio

Kömür sobası ve doğal gaz kaynaklı karbonmonoksit zehirlenmelerinin karşılaştırılması ve Nötrofil/Lenfosit oranının değerlendirilmesi

Yahya Kemal Günaydın[1], Kubilay Vural[2], Mehtap Akçil Ok[3], Yavuz Katırcı[4], Dilber Üçöz Kocaşaban[4], Yavuz Yiğit[5], Figen Coşkun[6]

ABSTRACT

Objective: The aim of our study is to present the epidemiologic, clinical, laboratory and prognosis differences between the coal stove origin poisoning and natural gas leakages. We also aimed to investigate relationship between the severity of clinical picture, prognosis, complications develop in CO poisoning with neutrophil/lymphocyte ratio (NLR) at the initial admission.

Methods: All the acute carbon monoxide cases who applied to Ankara Training and Research Hospital Emergency Medicine Clinic between October 2009 and April 2010 were included to this prospective study. CO poisoning diagnosis was made by the history of CO poisoning with carboxyl hemoglobin (COHb) concentration is over 10%. 100 patients were included to our study.

Results: Of the patients, 55(55%) were poisoned from the coal-stove and 45(45%) from natural gas leakage. The mean COHb level of the natural gas group was significantly high (p=0.01). The mean value of GCS of the natural gas group was significantly lower (p=0.018). The number of patients with indication for HBO therapy were 17 and 6 in the natural gas group and coal-stove group, respectively, being significantly higher in the natural gas group(p=0.001). There was no statistically significant relationship between the value of NLR and values of COHb, troponin, and GCS (p=0.872, p=0.470, and p=0.896, respectively).

Conclusions: Carbon monoxide poisoning from natural gas leakage is more toxic than that from the coal-stove. There is no relationship between NLR at the time of presentation and the severity of clinical findings, prognosis and complications.

Key words: Carbon monoxide poisoning, coal-stove; natural gas, Neutrophil/lymphocyte ratio

ÖZET

Amaç: Bizim çalışmamızdaki amacımız ülkemizde karbonmonoksit (CO) zehirlenmesinin en sık nedeni olan kömür sobasından kaynaklanan zehirlenmeler ile doğal gaz kaçaklarının yol açtığı zehirlenmeler arasındaki epidemiyolojik, klinik, laboratuar ve prognoz farklılıklarını ortaya koymaktır. Ayrıca CO zehirlenmelerinde oluşan klinik tablonun şiddeti, prognozu, komplikasyonları ile başvuru sırasındaki nötrofil/lenfosit oranı (NLO) arasındaki ilişkiyi incelemeyi amaçladık.

Yöntemler: Bu prospektif çalışmaya Ankara Eğitim ve Araştırma Hastanesi Acil Tıp Kliniği'ne 2009 Ekim – 2010 Nisan tarihleri arasında başvuran akut karbonmonoksit zehirlenmesi olgularının tümü alındı. CO zehirlenmesi tanısı, anemnezinde CO zehirlenmesine neden olabilecek öykünün olması ile birlikte karboksihemoglobin (COHb) konsantrasyonunun % 10'nun üstünde olması şeklinde konuldu. Çalışmamıza toplam 100 hasta dahil edildi.

Bulgular: 55 (%55) hasta kömür sobasıyla zehirlenirken, 45 (%45) hasta doğal gaz kaynaklı zehirlenmişti. Doğal gazdan zehirlenen grubun ortalama COHb düzeyi anlamlı bir şekilde daha yüksekti (p=0,01). Doğal gaz grubunun ortalama Glaskow koma skoru (GKS) değeri anlamlı bir şekilde daha düşüktü (p=0,018). Doğal gazdan zehirlenen grupta 17 hastanın HBO (hiperbarik oksijen) tedavisi ihtiyacı olurken, kömür sobasından zehirlenen grupta ise sadece 6 hastanın HBO tedavisi ihtiyacı oldu. Doğal gazdan zehirlenen grupta kömür sobasından zehirlenen gruba göre anlamlı bir şekilde HBO tedavisi ihtiyacı daha fazlaydı. (p=0,001) NLO ile COHb oranı, troponin ve GKS değerleri arasında istatistiki olarak anlamlı bir ilişki bulunmadı (Sırasıyla; p=0,872, p=0,470, p=0,896).

Sonuç: Doğal gaz kaynaklı CO zehirlenmeleri kömür sobası zehirlenmelerine göre daha toksik olabilmektedir. CO zehirlenmelerinde oluşan klinik tablonun şiddeti, prognozu, komplikasyonları ile başvuru sırasındaki NLO arasında her hangi bir ilişki yoktur.

Anahtar kelimeler: Karbonmonoksit zehirlenmesi, kömür sobası, doğal gaz, Nötrofil/Lenfosit oranı

[1] Konya Training and Research Hospital, Department of Emergency Medicine, Konya, Turkey
[2] Ordu State Hospital, Department of Emergency Medicine, Ordu, Turkey
[3] Başkent University, Faculty of Science and Letters, Statistics and Computer Science, Ankara, Turkey
[4] Ankara Training and Research Hospital, Department of Emergency Medicine, Ankara, Turkey
[5] Kocaeli Derince Training and Research Hospital, Department of Emergency Medicine, Kocaeli, Turkey
[6] Kırıkkale University, Faculty of Medicine, Department of Emergency Medicine, Kırıkkale, Turkey

Yazışma Adresi /Correspondence: *Yahya Kemal Günaydın,*
Konya Training and Research Hospital Department of Emergency Medicine, Konya, Turkey Email: gsykg@yahoo.com
Geliş Tarihi / Received: 14.07.2015, Kabul Tarihi / Accepted: 11.08.2015

INTRODUCTION

Carbon monoxide (CO) is a colorless, odorless, tasteless, and non-irritating gas. Carbon monoxide poisoning is one of the most common causes of poisoning worldwide, and the very first cause of fatal suicides and fatal accidental poisonings [1,2].

Carbon monoxide is a product of incomplete combustion of hydrocarbon fuels [3]. It is often produced by incomplete combustion of wood, coal and other fuels used in heaters such as stoves, boilers, combination boilers, and fireplaces, or by the exhaust smoke of motor vehicles and generators in closed areas that are poorly aerated. Furthermore, fires, vehicles using natural gas, paint thinner, and spray paints may cause CO poisoning [4].

Carbon monoxide rapidly combines with hemoglobin (Hb) to form carboxyhemoglobin (COHb) by readily displacing the oxygen (O2) in the Hb. Carboxyhemoglobin prevents hemoglobin from releasing oxygen in tissues, effectively reducing the oxygen-carrying capacity of the blood, and consequently leading to tissue hypoxia. The affinity of Hb to CO is 210 times higher than its affinity to O_2 [5,6]. The reason for CO toxicity may be considered as a competition between CO and O_2 for Hb, but its basic mechanism is a combination of tissue hypoxia and cellular damage directly caused by CO [7].

The symptomatology may differ in individuals exposed to CO under the same conditions. Many individuals remain asymptomatic when the level of COHb is under 10%. A COHb level of under 20% generally causes nausea, headache, and mild dyspnea; a level between 20-40% leads to vomiting, loss of judgment, and blurred vision, and a level above 40% causes ataxia, confusion, syncope, coma, and tachypnea. COHb levels are not prognostic, and deaths have been reported even with quite low levels [8]. After many symptom-free days, about 12% of the patients may demonstrate neurological sequelae such as memory loss, personality changes, demantia, psychosis, cerebellar ataxia, and parkinsonism [6,9]. Angina, pulmonary edema, arrhythmia, and ischemic ECG changes may be observed in particularly prone and comorbid patients [10].

The purpose of this study was to determine the epidemiological, clinical, laboratory, and prognostic differences between CO poisoning from coal-stoves, which is the most frequent cause of CO poisoning in Turkey, and CO poisoning from natural gas leakage [8]. Depression of the central nervous system, cardiac complications, and enzyme changes may develop in CO poisoning. When these symptoms are caused by reasons other than CO poisoning, they show a relationship with neutrophil/lymphocyte ratio (NLR) [11-15]. Based on this relationship, we aimed at assessing the relationship between NLR at the time of presentation and the severity of clinical findings, cardiac and neurological complications.

METHODS

Study Design and Patient Population

This prospective study included all cases of acute CO poisoning from coal-stove or natural gas leakage presenting to the Emergency Clinic of Ankara Training and Research Hospital, Ankara, Turkey, between October 2009 and April 2010. Cases with recent acute infection or inflammatory disease and cases who not arrive within 30 minutes were excluded from the study. The criteria for the diagnosis of CO poisoning were patient history and a COHb concentration of over 10%. Upon admission, physical and neurological examinations of the patients were performed and the Glasgow Coma Scale (GCS) was assessed. The possibility of myocardial ischemia or necrosis was checked with electrocardiography and troponin values. Furthermore, complete blood count and biochemical parameters were assessed routinely in all patients.

All patients received oxygen therapy with mask in the observation room. Some patients received normobaric oxygen (NBO) therapy, while others were transferred to another center for hyperbaric oxygen (HBO) therapy. The patients sent to the other centers returned to our hospital for follow-up after completing their HBO therapy. The indications for HBO therapy were presence of a GCS of under 8, neurological deficit, myocardial injury or ischemia, history of syncope or presenting with syncope, and pregnancy [16,17]. The HBO was administered at 2.8 bar atmospheric and oxygen pressure for 90 minutes.

The study comprised a total of 117 patients. Of these patients, 13 were excluded from the study because they had a COHb concentration of lower than 10% and another 4 were further excluded because they interrupted their therapy by leaving the hospital without informing us. The study protocol was approved by the Hospital Ethics Committee, and written and signed consent was obtained from all patients prior to the study.

Statistical analysis

The statistical analyses were made using the SPSS 15.0 (SPSS Inc, Chicago, Illinois) software. The quantitative variables were expressed as mean ± standard deviation and categorical variables such as number of cases (%). The normal and the abnormal distributions of two groups were assessed using the "Kolmogorov Smirnov test". For comparison of the differences between the groups, the independent-t test was used for the quantitative variables and the chi-square test was used for the categorical variables. The relationship between NLR and values of COHb level, GCS and troponin examined with Pearson Correlation test. The statistical significance was a p value of under 0.05 with a 95% confidence.

RESULTS

Demographic data

Of the patients, 36 (36%) were males and 64 (64%) females, and the mean age was 33.94±14.07 year. Only 1 patient (1%) had poisoned herself in the act of suicide; the rest of the cases were accidental. Of the patients, 55 (55%) were poisoned from the coal-stove and 45 (45%) from natural gas leakage. The natural gas group and coal-stove group were similar to each other in terms of age and gender (p=0.311 and p=0.738, respectively) However, CO exposure time were statistically significantly higher in coal-stove group. (p<0.001) (Table 1).

Table 1. Demographic data of the patients

		Coal Stove Group (n=55)	Natural Gas Group (n=45)	p
Gender				
	Female, n (%)	36 (56.3)	28 (43.8)	0.738[a]
	Male, n (%)	19 (52.8)	17 (47.2)	0.738[a]
Age (year)		35.23±13.98	32.35±1416	0.311[b]
CO exposure time (minute)		351.27±256.95	146.89±125.91	<0.001[b]

[a] Chi-square test, [b] Independent T test

Seventy-three (73%) of the patients had presented with non-specific symptoms such as headache, dizziness, and nausea-vomiting. The first electrocardiographic examination at the time of presentation revealed normal sinus rhythm in 77 (77%) patients. Seventy-seven (77%) were treated with NBO and 23 (23%) with HBO. The patients' mean values for COHb level, NLR, and GCS were % 28.63±10.53, 3.58±2.27, and 14.16±10.53, respectively (Table 2). Four (4%) patients required endotracheal intubation and mechanical ventilation. Only one (1%) patient died.

Primary Results

The mean COHb levels in the natural gas group and coal-stove group were 31.70±11.73% and 26.13±8.76%, respectively. The mean COHb level of the natural gas group was significantly high (p=0.01). The mean values of GCS in the natural gas and coal-stove groups were 13.47±3.15 and 14.73±1.63, respectively. The mean value of GCS of the natural gas group was significantly lower (p=0.018). The number of patients with indication for HBO therapy were 17 and 6 in the natural gas group and coal-stove group, respectively, being significantly higher in the natural gas group (p=0.001). There was no significant difference between the two groups in terms of troponin values (p=0.21).

Secondary results

There was no statistically significant relationship between the value of NLR and values of COHb, troponin, and GCS (Table 3; p=0.872, p=0.470, and p=0.896, respectively). The NLR values in the coal-stove and natural gas groups were 4.42±2.29 and 2.55±1.79, respectively, being significantly higher in the coal-stove group (Table 4, p=0.001).

Table 2. Patients' features

Symptoms on admission		n (%)
	Headache, dizziness, vomiting	73 (73)
	Confusion, unconsciousness,seizures	18 (18)
	Palpitation, chest pain	5 (5)
	Syncope	4 (4)
First electrocardiography (ECG)		
	Normal sinus rhythm	74 (74)
	Sinus tachycardia	15 (15)
	ST segment depression	8 (8)
	Sinus arrhythmia	2 (2)
	ST segment elevation	1 (1)
Indications for HBO therapy		
	Unconsciousness	9 (9)
	Unconsciousness + neurological deficit	2 (2)
	Unconsciousness + myocardial ischemia	4 (4)
	Unconsciousness + pregnancy	1 (1)
	Myocardial ischemia	4 (4)
	Pregnancy	3 (3)
	No indication	77 (77)
Laboratory		
	NLR	3.58±2.27
	COHb level (%)	28.63±10.53
	Troponin (mg/dl)	0.04±0.14
Glasgow Coma Scale		14.16±10.53

Values are expressed as mean ± standard deviation or number of patients (%).

Table 3. The relationship between NLR and values of COHb level, Glasgow Coma Scale (GCS) and troponin

	GCS	COHB (%)	Troponin (mg/dl)
NLR	r=0.013, p=0.896	r=-0.016, p=0.872	r=-0.073, p=0.470

Pearson Correlation test

Table 4. Comparison of coal stove group with natural gas group

	Coal Stove (n=55)	Natural Gas (n=45)	p
COHb level (%)	26.13±8.76	31.70±11.73	0.01[b]
NLR	4.42±2.29	2.55±1.79	0.001 [b]
GCS	14.73±1.63	13.47±3.15	0.018 [b]
Troponin (mg/dl)	0.03±0.06	0.063±0.19	0.21 [b]
Patients with indication for HBO therapy, (n)	6	17	0.001 [a]

HBO: Hyperbaric oxygen, [a]Chi-square test, [b]Independent T test

DISCUSSION

We have concluded that CO poisoning from natural gas is more toxic than that from the coal-stove, because the mean COHb values and the number of HBO therapy-indicated patients were significantly higher in the natural gas group than those in the coal-stove group. Moreover, the mean GCS in the natural gas group at the time of presentation was

significantly lower that that in coal-stove group. However, when the troponin values on admission was considered as a marker of cardiac injury, there was no difference between the two groups. These results cannot be compared with the literature, because as far as we know, this study is the first and only study on the subject.

The second purpose of this study was to assess the relationship between NLR at the time of presentation and the severity of clinical findings and cardiac and neurological complications in cases of CO poisoning. No significant relationship was found between them. There was no significant relationship between NLR and values of COHb, troponin, and GCS either. Although NLR in the coal-stove group was significantly higher than that in the natural gas group, we concluded that this situation was of no significance.

Various studies have reported that NLR is associated with central nervous system depression, cardiac complications, and enzyme changes seen in CO poisoning [11-15]. It has been also reported that the high value of NLR is associated with short- or long-term mortality in patients with acute myocardial infarction [11,12]. Kyne et al. [18] followed-up 185 patients with acute myocardial infarction and found a significant relationship between cardiac insufficiency and high NLR at the time of admission. Gazi et al. [19] have reported that high NLR in patients with myocardial infarction is associated with development of complications. In our study, we found no significant relationship between NLR and troponin values at the time of presentation.

Yardan et al. [20] have demonstrated that the levels of serum neuron-specific enolase and S100B protein in CO poisoning are associated with the patients' state of consciousness. We considered the same situation for NLR, but with no result. However, Ertaş et al. [21] found NLR to be significantly high in the patient group with thromboembolic paralysis in their study on patients with non-valvular atrial fibrillation. However, we have come to the conclusion that NLR is not associated with depression of the central nervous system in cases of CO poisoning.

The main limitation of our study was that the study was single-centered and comprised a limited number of cases. Another limitation was that the ECG was performed and troponin values were determined only at the time of the patients' presentation, and not repeated afterwards.

In conclusion, CO poisoning from natural gas leakage is more toxic than that from the coal-stove. There is no relationship between NLR at the time of presentation and the severity of clinical findings, prognosis and complications. A higher number and multi-centered studies on the subject are required to validate these findings.

REFERENCES

1. Raub JA, Mathiue-Nolf M, Hampson NB, et al. Carbon monoxide poisoning-a public health perspective. Toxicology 2000;145:1-14.
2. Turan M.İ, Çayır A, Olgun H. Delayed encephalopathy after acute carbon monoxide poisoning. Dicle Med J 2014;41:217-218.
3. Omaye ST. Metabolic modulation of carbon monoxide toxicity. Toxicology 2002;180:139-150.
4. Abelsohn A, Sanborn MD, Jessiman BJ, et al. Identifying and managing adverse environmental health effects: 6. Carbon monoxide poisoning. CMAJ 2002;166:1685-1690.
5. Ernst A, Zibrak JD. Carbon monoxide poisoning. N Engl J Med 1998;339:1603-1606.
6. Turner M, Hamilton-Farrell MR, Clark RJ. Carbon monoxide poisoning: an update. J Accid Emerg Med 1999;16:92-96.
7. Raphael JC, Elkharrat D, Jars-Guincestre MC, et al. Trial of normobaric and hyperbaric oxygen for acute carbon monoxide intoxication. Lancet 1989;2:414-419.
8. İnal V. Carbon monoxide poisoning and treatment. Turkiye Klinikleri J Anest Reanim 2005;3:34-41.
9. Choi IS. Carbon monoxide poisoning: systemic manifestations and complications. J Korean Med Sci 2001;16:253-261.
10. Handa PK, Tai DY. Carbon monoxide poisoning: a five year review at Tan Tock Seng Hospital, Singapore. Ann Acad Med Singapore. 2005;34:611-614.
11. Horne BD, Anderson JL, John JM, et al. Which white blood cell subtypes predict increased cardiovascular risk? J Am Coll Cardiol 2005;45:1638-1643.
12. Núñez J, Núñez E, Bodí V, et al. Usefulness of the neutrophil to lymphocyte ratio in predicting long-term mortality in ST segment elevation myocardial infarction. Am J Cardiol 2008;101:747-752.
13. Macrez R, Ali C, Toutirais O, et al. Stroke and the immune system: from pathophysiology to new therapeutic strategies. Lancet Neurol 2011;10:471-480.
14. Nagai M, Terao S, Yilmaz G, et al. Roles of inflammation and the activated protein C pathway in the brain edema associated with cerebral venous sinus thrombosis. Stroke 2010;41:147-152.

15. Kamisli S, Kamisli O, Gonullu S, et al. The prognostic value of increased leukocyte and neutrophil counts in the early phase of cerebral venous sinus thrombosis. Turk J Cerebrovasc Dis 2012 18:2;39-42.

16. Hampson NB, Mathieu D, Piantadosi CA, et al. Carbon monoxide poisoning: interpretation of randomized clinical trials and unresolved treatment issues. Undersea Hyperb Med 2001;28:157-164.

17. Brvar M, Mozina H, Osredkar J, et al. S100B protein in carbon monoxide poisoning: a pilot study. Resuscitation 2004;61:357-360.

18. Kyne L, Hausdorff JM, Knight E, et al. Neutrophilia and congestive heart failure after acute myocardial infarction. Am Heart J 2000;139:94-100.

19. Gazi E, Bayram B. Relationship between Neutrophil/Lymphocyte ratio and complications in patients with acute myocardial infarction. Anatol J Clin Invest 2012;6:17-20.

20. Yardan T, Cevik Y, Donderici O. et al. Elevated serum S100B protein and neuron-specific enolase levels in carbon monoxide poisoning. Am J Emerg Med 2009;27:838–842.

21. Ertaş G, Sönmez O, Turfan M. et al. Neutrophil/lymphocyte ratio is associated with thromboembolic stroke in patients with non-valvular atrial fibrillation. J Neurol Sci 2013;324:49-52.