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Original Article

Case Report

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Editorial

Dear Readers,

We present to you the first issue of our journal for 2025. In this issue, we have published 2 original article and 2 case reports and and that we think you will read with pleasure and interest. We hope that your scientific support will continue to increase in 2025. We would like to thank everyone who contributed to our journal for their support and contributions.

Best Regards.

Eurasian Journal of Toxicology Editorial Board

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Eurasian Journal of Toxicology

Evaluation of Carbon Monoxide Poisonings Observed After the February 6, 2023 Kahramanmaraş Earthquake

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Abstract

Objective: Carbon monoxide poisoning is a significant public health concern that can increase following natural disasters. This study aims to determine the frequency, etiological causes, and clinical characteristics of carbon monoxide poisoning cases presenting to the emergency department in the post-earthquake period following the Kahramanmaraş earthquakes on February 6, 2023.

Materials and Methods: This study includes the demographic and clinical data of patients diagnosed with carbon monoxide poisoning who presented to the emergency department following the earthquake. Patients' age, gender, presenting symptoms, carboxyhemoglobin levels, treatment approaches, and clinical outcomes were recorded. The data were analyzed by comparing the pre-earthquake and post-earthquake periods.

Results: A significant increase in the number of patients presenting to the emergency department due to carbon monoxide poisoning was observed in the post-earthquake period. Most cases were associated with the use of heaters and generators in temporary shelter areas with inadequate ventilation. Clinically, the most common presenting symptoms were headache, dizziness, and altered consciousness. Additionally, the proportion of cases requiring hyperbaric oxygen therapy was found to have significantly increased.

Conclusions: A significant increase in the incidence of carbon monoxide poisoning was observed following the Kahramanmaraş earthquakes. This finding highlights the importance of ensuring safe heating methods and raising public awareness in the post-disaster period. Preventive strategies aimed at reducing carbon monoxide exposure should be developed as part of disaster management.

Keywords: Carbon Monoxide Poisoning, Earthquake, Disaster, Carboxyhemoglobin

Introduction

Carbon monoxide (CO) poisoning remains a significant public health issue worldwide, being a preventable yet potentially severe cause of morbidity and mortality[1]. CO is a colorless, odorless, and tasteless gas, making it difficult to detect. When inhaled, it rapidly binds to hemoglobin, forming carboxyhemoglobin (COHb), which reduces oxygen-carrying capacity, leading to tissue hypoxia and metabolic disturbances. CO poisoning can present with a wide range of clinical manifestations, including headache, dizziness, altered consciousness, as well as cardiovascular and neurological complications, and can be fatal in severe cases[2]. Carbon monoxide (CO) poisoning can lead to clinical findings such as hypothermia, erythematous (red) skin changes, and bullae formation in pressure-sensitive areas. Oxygen is administered as an antidote in treatment[3]. The most common causes of CO exposure include heaters, stoves, water heaters, and generators that operate with incomplete combustion of fossil fuels. The use of these devices, especially in poorly ventilated conditions, can result in CO accumulation, increasing the risk of poisoning. After natural disasters, particularly large-scale catastrophes such as earthquakes, the risk of carbon monoxide (CO) poisoning tends to increase. The primary reasons for this include restricted access to safe heating and energy sources due to infrastructure damage, the use of inappropriate heating systems in temporary shelter areas, and prolonged stays in enclosed spaces. The literature reports a significant rise in CO poisoning cases following the 1999 Marmara Earthquake and the 2011 Japan Earthquake. These events highlight the impact of CO exposure on public health in the aftermath of natural disasters and emphasize the importance of preventive strategies[4,5].

Köseoğlu et al. emphasized that major disasters like earthquakes not only cause traumatic injuries but also lead to significant secondary health issues. Their study highlighted the burden on healthcare systems, particularly due to conditions such as crush syndrome and rhabdomyolysis. Similarly, our study demonstrates a marked increase in carbon monoxide poisoning cases post-earthquake, underscoring the need for a comprehensive approach to post-disaster health risks[6].

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The earthquakes centered in Kahramanmaraş, Turkey, on February 6, 2023, caused widespread destruction and affected millions of people, leading to significant challenges in shelter, heating, and energy supply. In the post-disaster period, thousands of people were forced to live in temporary shelters such as tents and containers, resulting in the widespread use of unsafe heating methods. Following this disaster, which occurred during the winter months, the improper use of stoves, catalytic heaters, and generators in enclosed spaces may have contributed to an increase in carbon monoxide poisoning cases.

This study aims to evaluate the frequency, etiological causes, and clinical characteristics of carbon monoxide poisoning cases in the post-earthquake period following the Kahramanmaraş earthquakes on February 6, 2023. By comparing the pre-earthquake and post-earthquake periods, the study seeks to identify changes in CO exposure. The findings obtained will contribute to the development of public health policies in the aftermath of disasters and the strengthening of strategies to prevent carbon monoxide poisoning.

Materials and Methods

This study was conducted using a retrospective observational design. The study included and compared patients who presented to the Emergency Department of Kahramanmaraş Sütçü İmam University with suspected carbon monoxide poisoning between February 6, 2022 – May 6, 2022, and February 6, 2023 – May 6, 2023.

Ethical Approval:
The study was approved by the Ethics Committee of Kahramanmaraş Sütçü İmam University with the decision dated 25.11.2024 and numbered 2024/31.

Inclusion Criteria:
- Age 18 years or older,
- Presence of symptoms consistent with CO exposure,
- Elevated carboxyhemoglobin (COHb) levels in arterial blood gas analysis.

Exclusion Criteria:
- Incomplete or insufficient medical records,
- Presence of alternative CO exposure sources, such as smoking,
- Low carboxyhemoglobin (COHb) levels in arterial blood gas analysis.

Data Collection:
Demographic information, clinical findings, laboratory results, and vital parameters were retrospectively obtained from the hospital automation system.

Table 1: Clinical and Laboratory Findings of Patients Based on COHb Levels

Parameter	Total Group (n=63)	COHb ≤ 15 (n=24)	COHb > 15 (n=39)	p Value
Age (years)	38,57 ± 14,20	37 ± 13	39 ± 15	0,625
pH	7,37 ± 0,05	7,39 ± 0,04	7,36 ± 0,05	0,043
pO2 (mmHg)	43,81 ± 36,09	59,3 ± 48,5	34,0 ± 20,8	<0,001
SaO2 (%)	94,24 ± 3,92	97 ± 3	93 ± 4	<0,001
Lactate (mmol/L)	2,19 ± 1,27	1,9 ± 1,4	2,4 ± 1,2	0,038
Reaspiratory Rate (breaths/min)	15,24 ± 2,73	14 ± 2	16 ± 3	<0,001
ED Stay (hours)	4,83 ± 1,63	4 ± 1	5 ± 2	0,062

Grouping:
Patients were categorized into two groups based on their COHb levels:
- **Group 1:** COHb ≤15 (n=24)
- **Group 2:** COHb >15 (n=39)

Statistical Analysis:
Data were analyzed using IBM SPSS v.23 software. Continuous variables were presented as mean ± standard deviation, while categorical variables were expressed as percentages (%). Appropriate non-parametric (Mann Whitney U) test were used for comparisons between groups. A p-value of <0.05 was considered statistically significant.

Results

A total of 63 patients were included in the study. Patients were categorized into two groups based on their COHb levels: Group 1 (COHb ≤ 15, n=24) and Group 2 (COHb > 15, n=39). Demographic data, arterial blood gas values, laboratory parameters, and vital signs are summarized in **Table 1**.

The graph presents the number of carbon monoxide poisoning cases in February, March, and April of 2022 and 2023. A significant increase in the number of cases is observed in February, March, and April 2023 compared to the corresponding period in 2022.

A significant increase in carbon monoxide poisoning cases was detected in the post-earthquake period, particularly in February. Additionally, it was observed that the majority of cases occurred during nighttime hours.

Discussion

This study demonstrates a significant increase in carbon monoxide (CO) poisoning cases following the Kahramanmaraş earthquakes on February 6, 2023. Major disasters such as earthquakes not only cause physical destruction but also lead to severe secondary public health

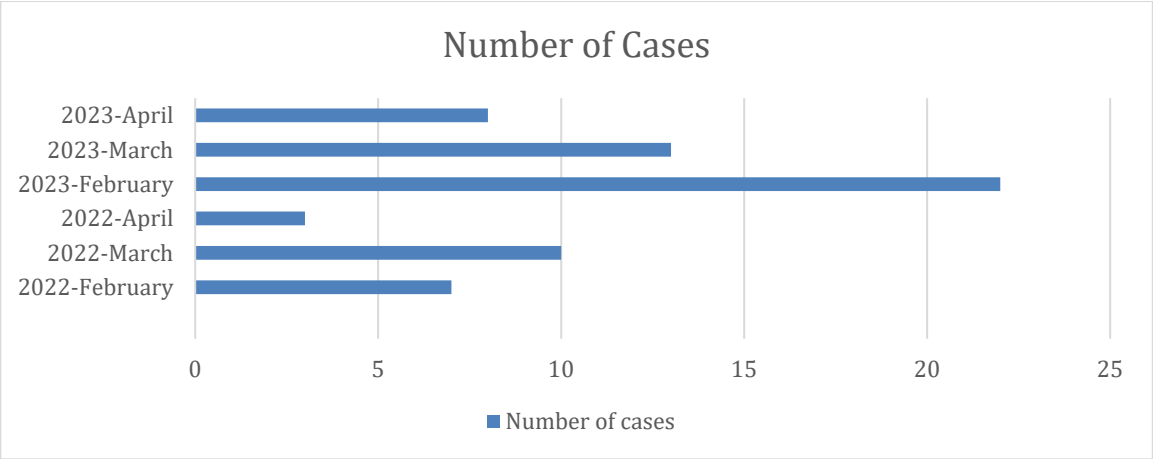

Figure 1: Monthly Distribution of Carbon Monoxide Poisoning Cases

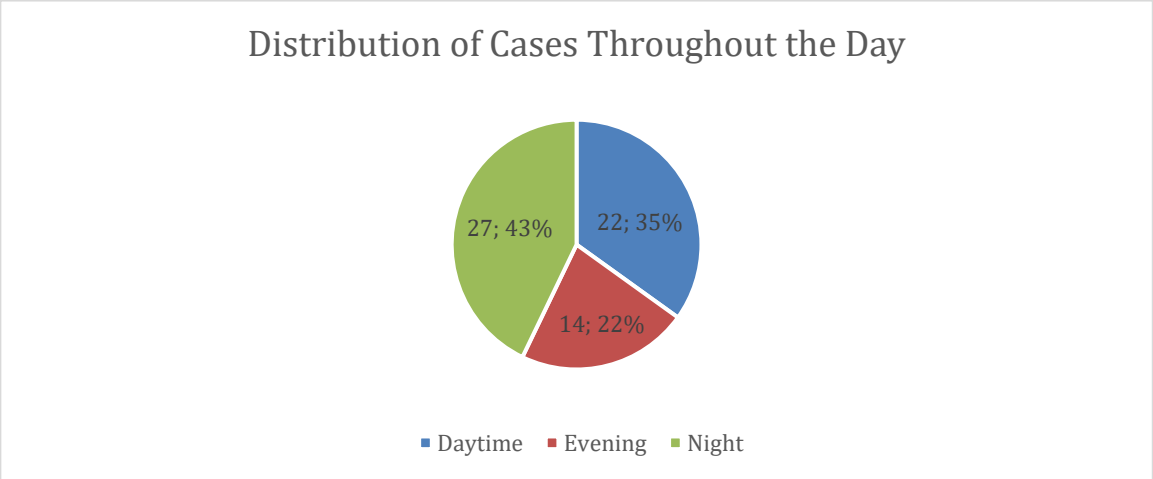

Figure 2: Distribution of Carbon Monoxide Poisoning Cases Throughout the Day. (43% of the cases occurred at night, 35% during the daytime, and 22% in the evening.)

issues1,2. In particular, the increased use of unsafe heating sources in temporary shelter areas has resulted in a notable rise in CO exposure-related poisonings4. Our findings indicate that this increase was especially pronounced during the cold winter months following the disaster and at nighttime. This observation is consistent with previous reports in the literature documenting a rise in CO poisoning cases after various disasters.

Previous research on post-disaster carbon monoxide poisoning has reported similar findings. For instance, a significant increase in CO poisoning cases was documented following the 1999 Marmara Earthquake and the 2011 Japan Earthquake. These studies highlighted that one of the primary risk factors was the use of stoves, charcoal grills, or other fossil fuel-powered devices in enclosed spaces by individuals residing in tents and containers to meet their heating needs after the earthquakes[5,7,8]. Similarly, our study also found that the majority of CO poisoning cases occurred during nighttime hours and that the use of fossil fuels in enclosed spaces was prevalent. Carbon monoxide (CO) is a toxic gas that is difficult to detect due to its colorless, odorless, and tasteless nature. It binds to hemoglobin with high affinity, forming carboxyhemoglobin (COHb), which prevents oxygen transport and leads to tissue hypoxia[9,10].

In our study, significant changes were observed in arterial blood gas parameters in patients with COHb > 15. Specifically, a decrease in pO_2, SaO_2, and pH levels was noted, while lactate levels showed a marked increase. These findings indicate that CO exposure leads to systemic hypoxia and triggers anaerobic metabolism. It is well established in the literature that carbon monoxide poisoning causes hypoxia and lactic acidosis, and the results of our study support this knowledge[11].

Additionally, our study identified a significant increase in respiratory rate associated with COHb levels. This finding suggests that CO exposure triggers a hypoxic respiratory response, indicating that patients compensate for hypoxia through physiological mechanisms. Previous studies have similarly demonstrated that CO exposure can lead to an increased respiratory rate[12]. This physiological response is

an important factor in understanding the clinical course of CO poisoning and should be considered as a key parameter in emergency department diagnosis.

The prevention of carbon monoxide poisoning is possible through early diagnosis and appropriate treatment approaches. In mild to moderate cases, high-flow oxygen therapy can reduce the half-life of COHb, while hyperbaric oxygen therapy is recommended for severe cases[13-15]. However, post-disaster conditions should be considered, as access to such treatments may be challenging. Therefore, the implementation of preventive measures is of critical importance. The observed increase in cases in our study further highlights the necessity of educating the public about carbon monoxide poisoning, ensuring safe shelter conditions, and promoting the use of carbon monoxide detectors in the aftermath of disasters.

One of the limitations of our study is its retrospective design. The retrospective collection of data may lead to missing clinical information. Additionally, it should be considered that the actual number of cases may be higher due to the overwhelming patient load. Nevertheless, our study presents valuable findings demonstrating the increased risk of carbon monoxide poisoning following a disaster and may serve as a guide for future research aimed at raising awareness on this issue.

Conclusion

This study demonstrates a significant increase in carbon monoxide poisoning cases following the Kahramanmaraş earthquakes on February 6, 2023. The majority of cases, which occurred particularly during the winter months and nighttime hours, were associated with temporary shelter conditions and the use of unsafe heating methods. Raising public awareness, promoting the widespread use of carbon monoxide detectors, and ensuring safe shelter environments are crucial for preventing CO poisoning in post-disaster periods. Additionally, healthcare professionals should be prepared for carbon monoxide poisoning cases during disaster periods. Future studies should focus on a more detailed assessment of risk factors in this field and the development of effective preventive strategies.

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Research Article

Eurasian Journal of Toxicology

Serum Creatine Phosphokinase as a Biomarker in Organophosphorus Poisoning

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Abstract

Background: Organophosphorous poisoning is a common problem in Nepal. Biochemical markers play an important role in the diagnosis and assessment of severity of Organophosphorous poisoning. Presently cholinesterase level which is an expensive biomarker is being used. However, new and cheaper biochemical markers are being studied. The objective of this study was to compare two laboratory biomarkers, creatine phospsokinase and Acetylcholine Esterase and its prognostic significance in Organophosphorous poisoning.

Methodology: A cross-sectional hospital-based study was conducted involving 40 patients with organophosphorus poisoning. Informed consent was obtained from caregivers, and patients of either sex who presented within 12 hours of ingestion/exposure were included. Cases of mixed poisoning, chronic alcoholism, liver/kidney disease, myositis, or use of medications (statins, fibrates, or steroids) were excluded from the study.

Clinical severity was categorized using the Peradeniya Organophosphorus Poisoning Scale. Venous blood samples were collected to measure serum creatine phospsokinase and Acetylcholine Esterase levels. Patients were treated with intravenous pralidoxime and atropine as per hospital protocol, avoiding intramuscular injections. After one week of admission, repeat serum creatine phospsokinase levels were re-measured.

The Pearson correlation coefficient was used to assess the relationship between Peradeniya Organophosphorus Poisoning score and creatine phospsokinase levels at admission, and the paired t-test was used to compare initial and final creatine phospsokinase and Acetylcholine Esterase levels, with a significance level of 0.05.

Results: Majority of patients enrolled in this study had mild Organophosphorous poisoning 32(80%) as per POP score whereas 6(15%) had moderate Organophosphorous poisoning and 2(5%) had severe poisoning. In patients with mild Organophosphorous poisoning the mean initial creatine phospsokinase level was 333.91±182.52 (IU/L). Patients with moderate Organophosphorous poisoning had a mean initial creatine phospsokinase level of 355.40±115.17(IU/L) where as in severe Organophosphorous poisoning the mean initial creatine phosphokinase level was 462.5±279.3(IU/L). The calculated Pearson correlation coefficient of Peradeniya Organophosphorus Poisoning with initial serum creatine phospsokinase level was 0.544 implying distinct positive correlation. The creatine phospsokinase levels in recovering patients showed a tendency to decrease, which was statistically significant in mild and moderate cases but not in severe cases.

Conclusions: Serum creatine phospsokinase level can be used as an alternative marker in the diagnosis and assessment of severity and prognostication in Organophosphorous poisoning especially in mild to moderated cases as shown in our study. Besides, the fall in the serum creatine phospsokinase level may also be used as marker of recovery.

Keywords: Organophosphorus Poisoning, Creatine phosphokinase, Acetyl Cholinesterase, Peradeniya Organophosphorus Poisoning Scale

Introduction

Acute poisoning by Organophosphorus pesticides (OP) is common in most parts of the developing world, particularly in Asia including Nepal where agriculture is the most common occupation in the country. The toxicity of OP compounds and the lack of appropriate medical facilities accounts for a high fatality rate. Easy accessibility of these pesticides has an important role in the choice of OP as a self-poison, and the incidence is particularly high among young people who are engaged in agriculture[1].

According to World Health Organization (WHO) one million serious unintentional poisonings occur annually, and an additional two million people seek hospital care for pesticide related suicide attempts[2]. Ravi et al reported in 2007 that the incidence of OP poisoning was around 126,000 over the period of 12 months in India[3]. In the year 1999-2000, 31% of all suicidal deaths in the country were due to poisoning[4]. Multicenter studies including five major hospitals across India in 1999- 2000 reported OP compounds were the commonest cause of poisoning, which comprised 52% of total cases[5].

A national study done in Bangladesh showed that self-poisoning caused 14 percent of all cause mortality among women of age group 10-50 years (3971 out of 28,998), with pesticides being the commonest[6]. The problem is particularly severe in Sri Lanka where pesticide poisoning was the commonest cause of hospital death in six rural districts during 1995[7]. The OP compounds had the largest

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burden of poisoning related morbidity and mortality in Nepal as shown in several hospital-based studies[5].

Organophosphate compounds inhibit the acetylcholinesterase enzyme (AchE) at muscarinic and nicotinic receptors. As a result, the patient develops symptoms like miosis, bradycardia, vomiting, profuse sweating, tachypnea, hypersalivation, lacrimation, altered sensorium, fasciculation, bronchospasm, blurred vision, photophobia, urination and defecation. If not treated promptly, patient can develop complications like respiratory paralysis, acute renal failure, seizures, arrhythmias, aspiration, coma and even death.[8]

Early recognition and timely intervention are of great importance to critical care providers and patients. OP toxicity is a clinical diagnosis which is confirmed by the measurement of cholinesterase activity. These investigations are not readily available everywhere. Although Red Blood Cell (RBC) and plasma (pseudo) cholinesterase (PChE) levels can both be used, RBC cholinesterase correlates better with CNS acetylcholinesterase activity and is, therefore, a more useful marker of OP poisoning. Erythrocyte cholinesterase is the more accurate of the two tests, however, plasma cholinesterase is easier to assay and is more widely available. Since RBC cholinesterase levels are not done in Nepal, plasma cholinesterase is the only option, which is not done in most laboratories and is an expensive investigation.[8]

There are novel alternatives to inexpensive and easily measurable biochemical markers of interest in OP poisoning, such as creatine phosphokinase (CPK), lactate dehydrogenase (LDH), serum immunoglobulins (IgG, IgA), and circulating complement components C3 and C4.

According to studies, OP poisoning is associated with elevated serum CPK levels, which could be used as a biomarker[8,9]. Hence this study was undertaken to see association and the prognostic significance of creatine phosphokinase in OP poisoning.

Methodology

Forty patients with OP poisoning were enrolled in this cross-sectional hospital-based study, conducted at Bir Hospital and SBH Army Hospital, Kathmandu, Nepal, over a period of 9 months after approval from Institutional Review Board. The patients were enrolled from the emergency department along with detailed history from the patient and/or caregiver. Confirmation of OP poisoning was done by label of packet/container of the poison consumed and serum Acetyl Choline Esterase (AChE) level at the time of admission. Informed consent was taken from the caregivers and patients were of either gender with history of ingestion or exposure to OP poison presenting to emergency department within 12 hours were included. However, patients who had mixed poisoning; chronic alcoholic intake, had history of chronic liver disease, chronic kidney disease or myositis; and patients taking any of medications including statins, fibrates, or steroids were excluded from the study Clinical severity was categorized according to Peradeniya Organophosphorus Poisoning (POP) scale as shown in Table 1.

Table 1: Peradeniya Organophosphorus poisoning (POP) Scale

Parameter	Criteria	Score
Pupil Size	≥ 2mm	0
	<2mm	1
	Pinpoint	2
Respiratory rate	<20/min	0
	≥ 20 min	1
	≥ 20 min with central cyanosis	2
Heart rate	>60/min	0
	41-60/min	1
	≤40/min	2
Fasciculation	None	0
	Present, generalized, continuous	1
	Both generalized/continuous	2
Level of consciousness	Conscious and rational	0
	Impaired response to verbal command	1
	No response to verbal command	2
Seizures	Absent	0
	Present	1

0-3: Mild poisoning, 4-7: moderate poisoning, 8-11: severe poisoning.

Venous blood sample was collected from a peripheral vein and sent for serum CPK and Acetyl Choline Esterase levels.

Patients were treated with intravenous PAM and atropine as per hospital protocol. Intramuscular injection was avoided in all the patients during the course of treatment.

At the end of one week from admission, the levels of serum CPK and serum AChE were measured.

Data was entered in Statistical Package of Social Science (SPSS) version 16 (SPSS Inc., Chicago IL, USA), and Microsoft Excel spreadsheet. Data analysis was done using SPSS (16 version) program and was depicted as tables and charts. Correlation of severity (POP score) with CPK level at admission was tested using Pearson correlation coefficient. The paired t test was used to analyze the difference between initial and final CPK and AChE levels with level of significance of 0.05.

Results

During the period of data collection of nine months (May 2013 to January 2014) there were a total of 40 patients enrolled into this study.

As shown in Figure 1, out of the 40 patients enrolled into the study 15(37%) were males and 25(63%) were females. The male: female ratio was 1: 1.7.

As illustrated in Figure 2, the majority of OP poisoning patients were in the age group ≥ 35 years which was 15 patients (37.5%). The next highest number of patients were in the age group 15-19 years where there were 12 (30%) patients. There were 7(17.5%) patients in the age group of 20– 24yrs. There were a similar number of patients in the age group 25- 29 years and 30-34 years 3 (7.5%).

The pie chart in Figure 3 shows that majority of patients enrolled in this study had mild OP poisoning patients. As per POP score, 32(80%) patients had mild OP poisoning, 6(15%) patients had moderate OP poisoning and 2 (5%) patients had severe poisoning.

The Table 2 shows that in patients with mild OP poisoning the mean initial AchE level was 2079.8±1361.17(IU/L). Patients with moderate OP poisoning had a mean initial AchE level of 525.83±133.45(IU/L) where as in severe OP poisoning the mean initial AchEe level was 147.0±93.33(IU/L). The mean initial AChE level was 1151.8±1131.4(IU/L) when all the patients were combined. This table shows that the POP score increases as the AChE levels decreases, which is illustrated in scatter diagram in Figure 4. The Pearson correlation coefficient was computed to be -0.576 indicating distinct negative correlation with p-value of <0.001.

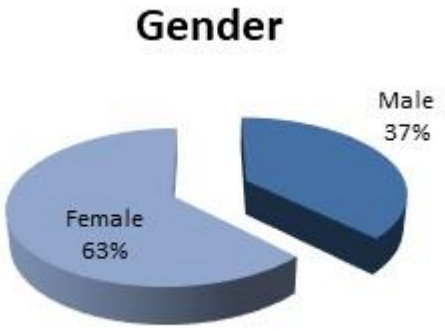

Figure 1: Sex distribution among OP poisoning patients

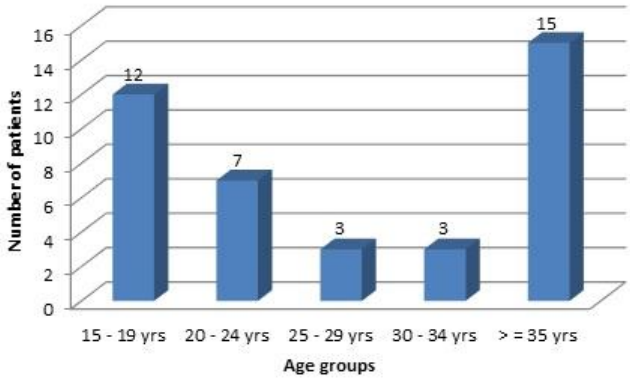

Figure 2: Age distribution among patients.

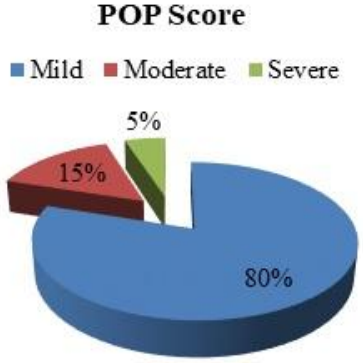

Figure 3: Severity of OP poisoning according to POP score

Table 2: Distribution of initial Ach E levels in relation POP score

POP score	Number of patients	Mean Initial AchE levels (IU/L)	± SD
Mild	32	2079.8	1361.17
Moderate	6	525.83	133.45
Severe	2	147.0	93.33
Total	40	1151.8	1131.47

In patients with mild OP poisoning, the mean initial CPK level was 333.91±182.52 (IU/L). Patients with moderate OP poisoning had a mean initial CPK level of 355.40±115.17(IU/L) where as in severe OP poisoning the mean initial CPK level was 561.25±60.85(IU/L). Table 3 shows that as the POP score increases the mean initial CPK level increases. Using scatter diagram as shown in Figure 5, Pearson correlation coefficient was calculated to be 0.544 implying distinct positive correlation.

The Table 4 shows that in patients with mild OP poisoning there is a reduction from the mean initial CPK level (333.91±182.52 U/L) to the mean final CPK level (61.59±17.30 U/L) which was statistically significant (p<0.001). In patients with moderate OP poisoning there is a reduction from the mean initial CPK level (355.40±115.17 U/L) to the mean final CPK level (75.16±28.62 U/L) which was statistically significant (p=0.015). However, in the group of patients with severe poisoning the reduction from the mean initial CPK level (561.25±60.85 U/L) compared to

Table 3: Distribution of initial CPK levels in relation POP score

POP score	Number of patients	Mean Initial CPK levels (IU/L)	± SD
Mild	32	333.91	182.52
Moderate	6	355.40	115.17
Severe	2	561.25	60.85

Table 4: Comparison of the initial and final CPK levels

POP score	Initial CPK (U/L)		Final CPK (U/L)		p value
	Mean	±SD	Mean	±SD	
Mild (0-3)	333.91	182.52	61.59	17.30	<0.001
Moderate (4-7)	355.40	115.17	75.16	28.62	0.015
Severe (8-11)	561.25	60.85	112.80	28.28	0.27

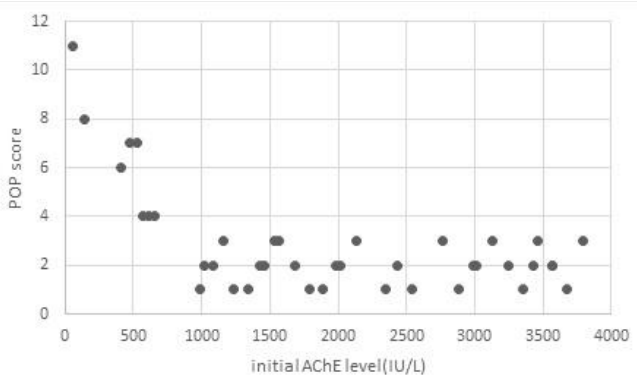

Figure 4: Severity of OP poisoning with initial AChE levels

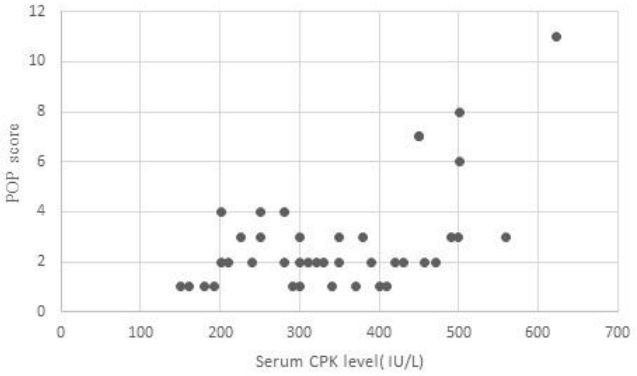

Figure 5: Scatter diagram showing correlation of severity of OP poisoning with CPK level

the mean final CPK (112.80±28.28 U/L) was statistically not significant (p=0.27).

Discussion

Organophosphate compounds are used for agriculture and industrial purposes and these compounds are easily available at a low cost. Poisoning with Organophosphates is a common cause of morbidity and mortality worldwide especially in South Asia. OP poisoning results in the inhibition of AChE at muscarinic and nicotinic receptors resulting in a range of symptoms. Complications of OP poisoning include acidosis, respiratory paralysis, renal failure, seizures, arrhythmias, aspiration, coma and even death. Early diagnosis is the key to cure. Till now estimation of serum cholinesterase and plasma cholinesterase levels have been used in the investigation and management of OP poisoning. However, these tests are costly and not done in most laboratories in the developing countries. Therefore, cheaper and easily available biochemical markers that can be used in OP poisoning are being studied. The aim of our study was to determine the association and prognostic significance of CPK in OP poisoning.

In our study majority of the enrolled patients were women (63%) vs men (37%). The male to female ratio was 1:1.7. This was similar to the gender distribution in the study by Hassan et al where they had majority of female patients.[10] However, in the study by Bhattacharya et al men comprised the majority (male: female=2:1).[8] In our study, age of the patients ranged from 15-56yrs. It was observed that most of the OP poisoning cases were in the higher age groups and in very young patients. In the study by Hassan et al, the age of patients enrolled ranged from 13-68 years and Bhattacharya et al enrolled patients from 16-44 years.[10]

Majority of patients enrolled in this study had mild OP poisoning 32 patients (80%) as per POP score. Out of 40 patients, 6 (15%) patients had moderate OP poisoning and 2 (5%) patients had severe poisoning. Bhattacharya et al who studied serum CPK as a probable marker of severity in OP poisoning had 32(50.8%) patients in the group of moderate OP poisoning, 27% in the mild group and 22.2% in the severe group.[8] Sen R et al who studied the prognostic biomarkers in Organophosphorus poisoning reported that as per the POP Score, 29 patients (23 females and 6 male) had mild poisoning, 45 had moderate poisoning (22 females and 23 males) and 26 (12 females and 14 males) had severe poisoning.[11]

In our study patients with mild OP poisoning had a mean initial AchE level of 2079.8±1361.17 (IU/L). Patients with moderate OP poisoning had a mean initial AchE level of 525.83±133.45 (IU/L) where as in severe OP poisoning the mean initial AchE level was 147.0±93.3(IU/L). The relationship between severity and AchE level was found to be statistically significant (p<0.001) with r value of -0.576 indicating distinct negative correlation. Determination of AChE and PChE level in blood has remained important for the initial screening of acute OP exposure which helps health-care professionals in early diagnosis and immediate treatment plan. Several studies have shown the relationship between cholinesterase levels and severity of OP poisoning and it has been used as a prognostic marker.[9,11,12]

In our study the patients with mild OP poisoning, the mean initial CPK level was 333.91±182.52 (IU/L). Patients with moderate OP poisoning had a mean initial CPK level of 355.40±115.17(IU/L) where as in severe OP poisoning, the mean initial CPK level was 561.25±60.85(IU/L). Our results showed that as the POP score increases, the CPK level increases as seen in the scatter diagram. Also, the Pearson correlation coefficient(r) was calculated to be 0.544 implying distinct positive correlation with p-value of <0.001. These findings were comparable to the findings of the study by Hassan NM et al. They reported that as the initial CPK level increased, the POP score also increased. (Mild – 89.1±27, Moderate- 273±96.7, Severe-688.8±86.7 U/L) which was statistically significant p<0.001).[10] The study by Sen R et al also reported that the correlation between severity of poisoning and serum CPK (Mild-449.65±325.4, Moderate-768.2±485.4, Severe-1324.74±141.6) showed a high degree of positive correlation (r = 0.625) and the correlation was also statistically significant (p =0.001).[11]

The study by Bhattacharya et al also showed a similar result. They also reported a positive correlation between OP poisoning severity and CPK levels. (Mild – 273.53±108.71, Moderate-456.06±77.20, Severe-1032.57±205.57 U/L) which was statistically significant (p<0.001). It was found that the mortality was more in patients with high initial CPK levels. Patients with severe poisoning have been shown to exhibit elevated levels of CPK. The presence of rhabdomyolysis in 'intermediate syndrome' is associated with increased CPK levels. The findings revealed that serum CPK levels are elevated in patients with severe organophosphate poisoning, even when intermediate syndrome is not present, likely due to muscle fiber necrosis observed in muscle biopsies.[8]

Our study showed, patients with mild OP poisoning had a reduction from the mean initial CPK level (333.91±182.52 U/L) to the mean final CPK level (61.59±17.30U/L) which was statistically significant (p<0.001). In patients with moderate OP poisoning, there was a reduction from the mean initial CPK level (355.40±115.17 U/L) to the mean final CPK level (75.16±28.62 U/L) which was also statistically significant (p<0.015). However, in the group of patients with severe poisoning the reduction from the mean initial CPK level (561.25±60.85 U/L) compared to the mean final CPK (112.80±28.28 U/L) was statistically not significant (p=0.27). Only two patients presented with severe OP poisoning. The decrease in CPK levels may have been statistically insignificant, attributable to the limited sample size within that group. Cases of severe OP poisoning necessitated a longer recovery period, resulting in a less consistent decline in CPK levels.

The CPK levels in recovering patients showed a tendency to decrease. Therefore, serial measurement of serum CPK level might be helpful in predicting the prognosis of OP poisoning.

In the study by Hassan NM et al, their comparison between initial and final CPK levels showed that there was a reduction in the final CPK levels with treatment in the mild and moderate cases which was statistically significant (p<0.001), while the changes among the severe group was not significant. They assumed that this was probably due to the widespread complication that occurred in the severe group in their study. These results were similar to our study.[10]

A more extensive study that encompasses a greater number of patients across each category (mild, moderate, and severe OP poisoning) will be beneficial in confirming our findings with increased reliability.

Conclusion

Serum CPK levels serve as a significant marker for assessing the severity of organophosphorus (OP) poisoning. This test is more accessible and cost-effective than measuring serum AChE levels. Consequently, serum CPK levels can be an invaluable resource for managing OP poisoning in resource-constrained areas. Furthermore, conducting serial CPK tests throughout the treatment process can aid in tracking the patient's recovery. Nonetheless, a key limitation is its non-specificity, making it essential to rule out other potential causes of elevated CPK levels.

Data availability

The datasets used in the study will be available from the corresponding authors upon reasonable request.

List of abbreviations

Ethics declarations

Ethical approval was taken from Institutional Review Board of National Academy of Medical Sciences, Kathmandu, Nepal.

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Competing interests

The authors declare no competing interests.

Consent for publication: Written informed consent was taken from the patients for the enrollment in study and publication of the article

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Case Report

Eurasian Journal of Toxicology

Can Tp-e/QTc Ratio and Blood Lactate Levels Serve as an Earlier Indicator Than Troponin for Detecting Cardiac Ischemia in Patients with Carbon Monoxide Poisoning in the Emergency Department?

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Abstract

Carbon monoxide (CO) poisoning is a clinical condition with serious cardiotoxic effects. This study reports two cases of CO poisoning to evaluate whether Tp-e/QTc ratio and blood lactate levels can serve as earlier indicators of cardiac ischemia than troponin levels. Two male patients, aged 37 and 47, who presented to the emergency department due to CO exposure, were analyzed. Their electrocardiographic (ECG) parameters, troponin levels, and lactate values were compared. Additionally, the clinical course of both patients was assessed.

In the first case (37 years old), ST depression in leads V1-V6 was detected on ECG, and troponin levels showed a progressive increase. Coronary angiography revealed no significant narrowing of the epicardial coronary arteries. The patient's QTc interval was 481 ms at admission, Tp-e duration was 79 ms, and Tp-e/QTc ratio was 0.16. Upon discharge, these values returned to normal. The second case (47 years old) had normal ECG and laboratory findings and was discharged without complications. Furthermore, in the first case, lactate levels were significantly higher at admission and remained elevated for a prolonged period.

Minimal QTc prolongation and a significant increase in lactate levels following CO poisoning may serve as early indicators of cardiac ischemia. Despite normal troponin levels, the first case required further cardiac evaluation. These findings suggest that monitoring QTc and lactate levels may help in the early detection of cardiac involvement in CO poisoning. However, further research is required to establish the prognostic value of these markers.

Keywords: Carbon monoxide poisoning, Cardiac ischemia, Tp-e/QTc ratio, Blood lactate level, Electrocardiography.

Introduction

Carbon monoxide (CO) poisoning can occur due to various sources, including fuels used for heating and cooking, industrial chemical production, poorly ventilated environments with motor vehicle emissions, leaks in home heating systems, and intentional exposure. Regardless of the source, CO exerts toxic effects on the human body, primarily due to tissue hypoxia, affecting the cardiovascular and neurological systems. Numerous studies in the literature have investigated the etiology, frequency, and prognostic factors of CO poisoning[1-4]. In CO poisoning, diagnosis is primarily based on a history of exposure, the presence of nonspecific symptoms, and blood carboxyhemoglobin (COHb) levels. However, there are a limited number of studies focusing on predicting prognosis and organ damage in emergency settings[5-7]. In this article, we present two cases in which we compare data suggesting that the cardiotoxic effects of CO poisoning may be detected earlier using specific parameters rather than troponin levels.

Case Presentations

Two patients presented to the emergency department approximately two hours after exposure to fumes from a coal-burning heating stove, complaining of headache, fatigue, nausea, vomiting, dizziness, and shortness of breath.

Case 1

A 37-year-old male patient admitted at the emergency department in stable condition with no impairment in consciousness. On physical examination, blood pressure, 138/88 mmHg; heart rate, 112 beats/min; body temperature, 36.4°C; and respiratory rate, 34/min. After presentation to the emergency department, 100% oxygen therapy was started at 15 L/min using a reservoir mask without respiration and intravenous access was provided. The patient did not report any active complaints other than feeling generally unwell. The patient had no medical history and tobacco use, prior surgical procedures, or regular medication use. On physical examination, breath sounds were normal, and there

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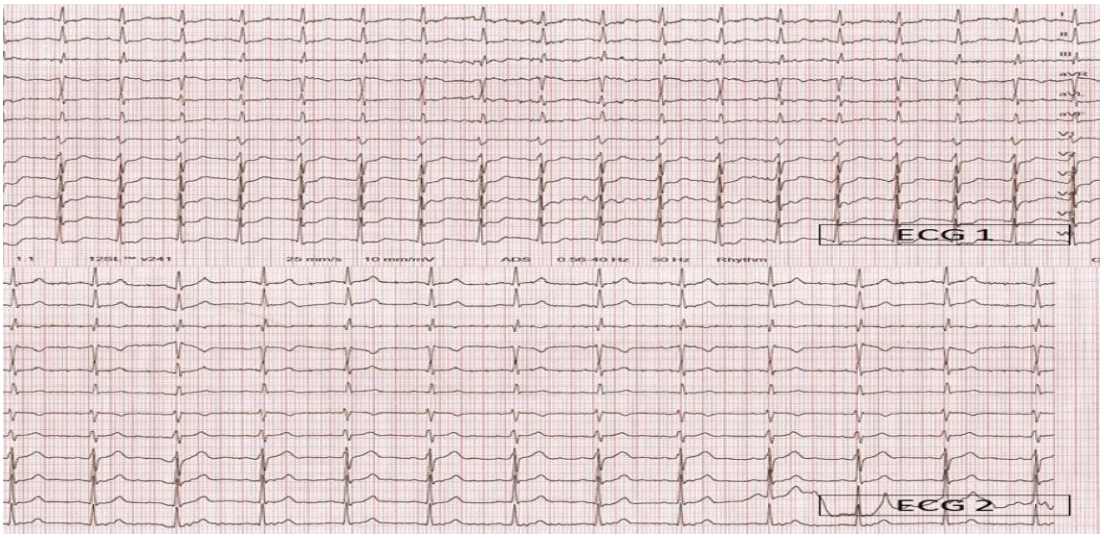

Figure 1: ECG 1-2

were no signs of dyspnea, neurological deficits, or chest pain. However, his electrocardiogram (ECG) revealed ST depression in leads V1-V6. Transthoracic echocardiography (TTE) showed no pathological findings, and chest radiography was unremarkable. At hospital admission, ECG findings (Figure 1, ECG 1) revealed a QTc of 481 ms, Tp-e of 79 ms, and a Tp-e/QTc ratio of 0.16. Upon discharge (Figure 1, ECG 2), QTc decreased to 456 ms, Tp-e was 80 ms, and the Tp-e/QTc ratio was 0.17. Additionally, lactate levels at admission were significantly higher in Case 1 compared to Case 2, and despite normobaric oxygen therapy, Case 1 exhibited prolonged lactate elevation relative to Case 2.

Due to the presence of ST depression in leads V1-V6 on initial ECG and progressive elevation of troponin levels, Case 1 was admitted to the coronary intensive care unit. As troponin levels continued to rise, the patient underwent coronary angiography (CAG), which revealed normal coronary arteries. Following 48 hours of observation, the patient was discharged after normalization of troponin levels and the absence of additional complications related to CO poisoning.

Case 2

A 47-year-old male patient, also in stable condition with no impairment in consciousness, presented with the following physical examination findings: blood pressure, 127/77 mmHg; heart rate, 98 beats/min; body temperature, 36.7°C; and respiratory rate, 32/min. He was immediately started on 100% oxygen therapy at 15 L/min via a non-rebreathing reservoir mask, and intravenous access was established. Like to Case 1, he had no medical history. He did not use tobacco, had no prior surgical history, and was not on any regular medication. His physical examination revealed normal breath sounds without signs of dyspnea, neurological deficits, or chest pain. His ECG and chest X-ray findings were within normal limits. Laboratory test results for both patients are presented in **Table 1**. Given he absence of active

Table 1: Patients' laboratory values

Patient	Laboratory values	1*	2**	3***	At discharge	Mean	Median	St. Deviation	Rate of Change	Normal values
Case 1	pH	7.26	7.35	7.41	7.36	7.34	7.35	0.06	0.008	7.35-7.45
	PO_2 mmHg	129	272	160	84	161.2	144.5	80.1	-3.75	83-108
	PCO_2 mmHg	27.5	38.4	31.5	39.4	34.2	34.9	5.6	0.99	32-48
	SO_2 %	98.2	99.4	99.2	96.3	98.2	98.7	1.4	-0.15	95-99
	Lac mg/dL	79	22	13	10	31	17.5	32.4	-5.7	5.0-14.0
	MetHb %	1.6	2.2	1.8	1.8	1,85	1.8	0.2	0.01	0-1.5
	O_2Hb %	67.4	67.4	96.3	96.5	81.9	81.8	16.7	2.4	94-98
	COHb %	29.8	11.9	1.3	0.9	10.9	6.6	13.5	-2.4	0.5-1.5
	$cHCO_3$ ST	14.5	20.6	22	22.4	19.8	21.3	3.6	0.65	-
	TROPONIN ng/L	300.5	777.4	1129	494	675.2	635.7	360.3	16.12	0-19.8
Case 2	pH	7.336	7.39	7.368	7.39	7.36	7.37	0.28	0.001	7.35-7.45
	PO_2 mmHg	208	247	128	81.1	166.2	168.5	75.0	-2.64	83-108
	PCO_2 mmHg	40.5	40	44	43.6	42.0	42.0	2.06	0.064	32-48
	SO_2 %	99.2	99.6	98.7	95.5	98.2	98.9	1.86	-0.077	95-99
	Lac mg/dL	27	9	7	7	12.5	8.0	9.7	-0.41	5.0-14.0
	MetHb %	2.1	1.5	2	1.7	1.8	1.8	0.27	-0.008	0-1.5
	O_2Hb %	69.9	96.1	95.7	92.7	88.6	94.2	12.5	0.47	94-98
	COHb %	27.2	2	1	1	7.8	1.5	12.94	-0.54	0.5-1.5
	$cHCO_3$ ST	23.1	24.2	24.2	25.7	24.3	24.2	1.06	0.54	-
	TROPONIN ng/L	10.7	34	65.9	91.6	50.5	49.9	35.5	1.68	0-19.8

*Admission to the hospital, **2nd Hour, ***6th Hour

complaints, normal laboratory values, and stable clinical findings, Case 2 was discharged 12 hours after admission.

Discussion

Pathological cardiac changes observed during CO poisoning are often associated with prognosis8. Electrocardiographic changes such as ST and T-wave abnormalities, QT prolongation, and arrhythmias are frequently reported in CO poisoning cases[8]. While a significant correlation between the cardiac effects of CO and T peak-to-T end (Tp-e) dispersion and Tp-e/QTc ratio has been documented, there is limited research on whether these parameters serve as early warning markers for cardiac ischemia in CO poisoning[9].

The slightly elevated QTc values observed in Case 1 align with existing literature suggesting that minimal QTc prolongation may serve as an early marker of cardiac involvement in CO poisoning9. Previous studies indicate that lactate levels rise before troponin in CO poisoning, suggesting that lactate may serve as an early biomarker for cardiac ischemia. Furthermore, lactate levels have been associated with prognosis prediction in CO poisoning cases[6,10].

In the present report, two cases with no significant differences in medical history were evaluated, yet Case 1 demonstrated a progressive increase in troponin levels compared to Case 2, raising concerns for acute coronary syndrome (ACS) and prompting the emergency department team to perform further investigation. Case 1 was monitored in the coronary intensive care unit (ICU), and subsequent coronary angiography (CAG) confirmed patent coronary arteries, thereby verifying cardiac involvement secondary to CO toxicity. Following comprehensive evaluation, treatment, and monitoring, the patient was discharged in good health. A retrospective comparison of the two cases was conducted to identify potential differences. To minimize measurement errors, ECG intervals (QT, QRS, and Tp-e) were manually measured using calipers and magnification. The QT interval was measured from the beginning of the QRS complex to the end of the T wave, and heart rate-corrected QT (QTc) was calculated using Bazett's formula. Tp-e interval was defined as the duration from the peak to the end of the T wave. Our findings support existing literature demonstrating the prognostic significance of elevated lactate in CO poisoning-related cardiac ischemia and hospital stay duration[6,10].

Limitations

One of the main limitations of this study is the lack of long-term follow-up for both cases, preventing further assessment of myocardial ischemia using imaging modalities such as myocardial scintigraphy. It is well established that age is a risk factor for poor prognosis in CO poisoning, partly due to the increased prevalence of comorbid conditions with aging. However, in our cases, the younger patient exhibited a more severe clinical course, suggesting that unidentified physiological factors may contribute to individual susceptibility to CO poisoning. These factors represent the primary limitations of the presented cases.

Conclusion

Both patients were exposed to CO in the same environment for the same duration. Despite no significant differences in baseline characteristics, one patient required advanced evaluation, treatment, and ICU admission. This case highlights the potential role of minimal deviations in QTc from normal values as a clinically relevant marker of coronary ischemia in CO poisoning.

Additionally, the persistently elevated lactate levels in Case 1, which demonstrated a weaker response to oxygen therapy, suggest that lactate measurements alongside QTc monitoring may provide valuable insights into the early cardiac effects of CO poisoning. These findings indicate that the combined assessment of these two parameters may improve early detection of cardiac involvement in CO poisoning. However, further research is required to establish rapid and reliable biomarkers that can be used at the time of emergency department admission to predict prognosis and reduce observation times in CO poisoning cases.

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Case Report
Eurasian Journal of Toxicology

A Colorful Complication Of Urinary Tract Infection: Purple Urine Bag Syndrome

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Abstract

Purple urine bag syndrome is a rare and distinctive manifestation of urinary tract infection that has been observed in patients with urethral catheterisation. The underlying mechanism of this phenomenon involves a reaction between the bacterial enzymes present in infected urine and the polyvinyl chloride components of the urine bag, leading to the manifestation of a distinctive purple colouration. The condition predominantly afflicts elderly individuals, particularly those with prolonged urethral catheterisation, and is more prevalent in the female demographic. The present case study focuses on an elderly male patient residing in a nursing home, who has a history of Alzheimer's disease and chronic kidney disease who was brought to the emergency department with complaints of oral intake disorder and constipation.

Keywords: Purple urine bag syndrome, urinary tract infection, urethral catheterisation

Introduction

In certain instances, as a consequence of urinary tract infection, the urine bag may exhibit a purple hue subsequent to the interaction of bacterial enzymes present in the urine with the urine bag. This phenomenon is designated as purple urine bag syndrome. Tryptophan, an essential amino acid, is ingested alongside food and subsequently metabolised to indole by bacterial enzymes present in the intestine. Subsequently, indole is transported to the liver, where it undergoes a transformation into indoxyl sulfate through a process of hepatic conjugation. Indoxyl sulfate is excreted in the urine and is degraded to indoxyl under alkaline conditions by bacteria producing sulfatase and phosphatase. Oxidation of indoxyl releases indigo (blue) and indirubin (red) pigments. When these pigments come into contact with the urinary catheter and urine bag containing polyvinyl chloride, a purple discolouration of the urine is observed[1]. Purple urine bag syndrome (PUBS) is usually seen in elderly patients who are followed with a long-term urinary catheter[2]. The primary risk factor for this condition is the presence of a long-term urinary catheter; other risk factors include female gender, constipation, recurrent urinary tract infections and dehydration[3,4]. In case of constipation, the time that food spends in the intestine increases, making it easier for bacteria to multiply and for tryptophan to be metabolized into indole.

The objective of this case report is to remind physicians of the rare and alarming nature of purple urine bag syndrome, emphasising that it can only be diagnosed by inspection.

Case Report

An 80-year-old male patient was brought to the emergency department with complaints of oral intake disorder and constipation. It was learned that the patient was residing in a nursing home and was being monitored with a urethral catheter for a long time. A review of the patient's medical history revealed the following: the patient had Alzheimer's disease, chronic kidney disease and an operation for inguinal hernia six years prior. A physical examination of the patient revealed a moderate general condition, with blurred consciousness, poor orientation, dehydration, and cachexia.

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Figure 1: Urine bag containing purple colored urine at the patient's emergency room admission

The Glasgow Coma Scale (GCS) score was 14 points. On inspection, purple colored urine was in the urinary bag (Figure 1). The patient's vital signs upon admission to the emergency department were as follows: Blood pressure: 87/56 mmHg, pulse rate: 105/min, body temperature: 36 °C, oxygen saturation: 99%, fingertype blood sugar: 127 mg/dL, Electrocardiography: normal sinus rhythm. The patient's blood sample exhibited the following laboratory parameters: pH: 7.10, HCO_3: 10.4 mmol/L, WBC: 8890 10^3/µL, Hgb: 13.2 g/dL, Plt: 230.000 / µL, Creatine: 9.62 mg/dL, BUN: 169 mg/dL, GFR: 5 ml/dk/1.73m^2, Na: 152 mmol/L, Cl: 118 mmol/L, K: 5.2 mmol/L, CRP: 28.7 mg/L, PCT: 0.54 µg/L. The patient's urine test results are as follows: pH: 8.5, protein: ++++, nitrite: negative, leukocyte: +++. Urinary ultrasound (USG) was scheduled to determine the underlying cause of the elevated creatinine levels. The results of the ultrasound scan revealed a heterogeneous hypoechoic appearance of approximately 63*48 mm in size, occupying the bladder lumen, suggesting the presence of a bladder tumor. Emergency hemodialysis was planned, and a temporary central catheter was inserted. PUBS was not a factor contributing to the development of acute kidney injury, but acute kidney injury and PUBS were a condition that occurred simultaneously. The urethral catheter was changed. Following the collection of urine and blood cultures, as a result of the questioning, it was learned that the patient had no allergy and empirical intravenous antibiotics (ceftriaxone 1 gr) were started. The hemodialysis procedure was conducted in the emergency room, and the patient was subsequently transferred to the intensive care unit for continued observation and treatment. However, during the patient's follow-up in the intensive care unit, his clinical condition continued to deteriorate and he died due to his other underlying diseases.

Discussion

PUBS is an atypical presentation of a urinary tract infection (UTI) characterized by the presence of purple coloration in the urine. The purpose of presenting this case is to remind physicians of the existence of purple urine bag syndrome, a rare condition that can be alarming to the unaccustomed eye. The etiology of PUBS is multifactorial, including factors such as female gender, prolonged use of urethral catheters, the presence of alkaline urine (which can stimulate the release of indoxyl sulfatase by bacteria), constipation (which can lead to increased colonic bacterial proliferation), and chronic renal failure[4,5].

Urinary tract infections are more prevalent among women than men due to anatomical differences, including a shorter urethra and its proximity to the vagina and anus. Purple urine bag syndrome, a condition associated with catheter-associated urinary tract infection, it is understandable that the female gender is more common in reported cases. When compared to existing literature, the patient in our case was male, and this condition is observed less frequently in male patients.

The geriatric population is particularly susceptible to constipation, a condition that is associated with a number of factors, including neurological diseases, prolonged immobility, and age-related changes in bowel function. Nutritional habits also play a role in the development of constipation. Constipation has been demonstrated to result in an increase in bacteria in the intestinal flora, which can lead to secondary infections. Constipation is a common symptom reported in patients with purple urine bag syndrome. Our case also exhibited constipation, chronic renal disease, and alkaline urine, findings that align with existing literature on the subject.

The increased frequency and duration of urethral catheter use in geriatric patients residing in nursing homes and hospitals may be a contributing factor to the development of urinary tract infections6, 7. Given the rising prevalence of catheter-associated urinary tract infections, a concomitant rise in reported cases of purple urine can be anticipated in forthcoming years.

Although purple urine bag syndrome is a clinical syndrome with a favorable prognosis, it is a clinical syndrome that should be considered due to the fact that the underlying causes and delayed treatment of urinary tract infections can be a significant cause of morbidity and mortality. In this particular case report, the patient succumbed to underlying diseases and exhibited an inadequate response to treatment, resulting in an exitus.

Conclusion

With this case report, purple urine bag syndrome, which can be diagnosed by inspection but is not encountered frequently, has been brought to the current literature data.

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