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Chest Pain in a Young Male with Carbon Monoxide Poisoning and Substance Abuse: A Case Report and Literature Review

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Case Report

Abstract

BACKGROUND: Carbon monoxide (CO) poisoning is the leading cause of poisoning-related deaths in the United States. In addition, myocardial infarction (MI) due to CO poisoning in a young, healthy adult is rare. On the other hand, smokeless tobacco, processed in various forms, is a controversial coronary heart disease (CHD) risk factor.

CASE REPORT: In this study, we describe a 29-year-old man who presented with acute chest pain following a night of smoking tobacco and using smokeless tobacco in the presence of carbon monoxide poisoning. ST-segment elevation was observed on an electrocardiogram, and echocardiography revealed akinesia. In addition, cardiac markers were elevated. In this particular instance, thrombolytic therapy demonstrated successful outcomes.

CONCLUSIONS: We believe the case and discussion could shed light on the emergency department management of such individuals. We advise clinicians to consider the possibility of coronary heart disease in carbon monoxide poisoning patients and to obtain a baseline electrocardiogram and cardiac markers.

Keywords: Chest pain, Carbon monoxide poisoning, Smokeless tobacco, Case report, Ischemic heart disease

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Introduction

Carbon monoxide (CO) poisoning is the leading cause of poisoning deaths in the United States. The incidence rate of CO poisoning in the US is 23.2 per 1 million of the population [1]. Neurological and cardiovascular manifestations are the most prevalent symptoms of CO poisoning. However, data regarding cardiovascular effects is largely unknown. More than one-third of patients with moderate and severe CO poisoning present with myocardial injury, as indicated by an elevation of cardiac markers or electrocardiogram (ECG) abnormalities [2].

Although some studies reported that CO poisoning patients presented with non-ST elevation myocardial infarction (NSTEMI) or, less frequently, ST-elevation myocardial infarction (STEMI), the patients described in these studies had risk factors for ischemic heart disease (IHD) that call into question the causality of CO poisoning [3-5]. The authors believe that STEMI is a rare phenomenon caused by CO poisoning in young, healthy individuals.

Smokeless tobacco, processed in various forms and most commonly consumed as moist snuff or locally known as *Nas*, is a controversial coronary heart disease (CHD) risk factor [6]. In addition, smokeless tobacco use is more prevalent among young adults in the Middle East, which may be attributable to its lower

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cost, less harmful appearance, reduced social stigma, and higher nicotine load [7].

In this study, we describe a case of CO poisoning in which a 29-year-old male presented to our emergency department for chest pain, as well as the associated diagnostic challenges.

Case Presentation

A 29-year-old male presented with persistent chest pain and was admitted to our emergency department eight hours after chest pain initiation. According to the patient, the chest pain was at its worst for the first four hours and remained the same for the following four hours. The patient reported that the chest pain began in the middle of the night while sleeping after smoking tobacco and ingesting the drug Nas. The patient had begun daily use of the Nas drug (a moist, powdered tobacco snuff) approximately one month prior. The patient was a construction worker staying in a room with a poor heating system and an elevated risk of carbon monoxide (CO) poisoning, as a young coworker had suffered from CO poisoning in the same room the previous night. Along with chest pain, the patient also experienced vertigo and lightheadedness. At presentation, vital signs were stable, and mild bibasilar lung rales were present. The ECG is illustrated in Figure 1.

Differential Diagnosis, Investigations, and Treatment

The ECG revealed a right bundle branch block with ST elevations of 1-2 mm in leads V1 and V2 and 1 mm diffusely. This ECG was compatible with epicardial coronary occlusion, pericarditis, myocarditis, and global myocardial ischemia due to hypoxemia. Due to the lack of coronary angiography and percutaneous

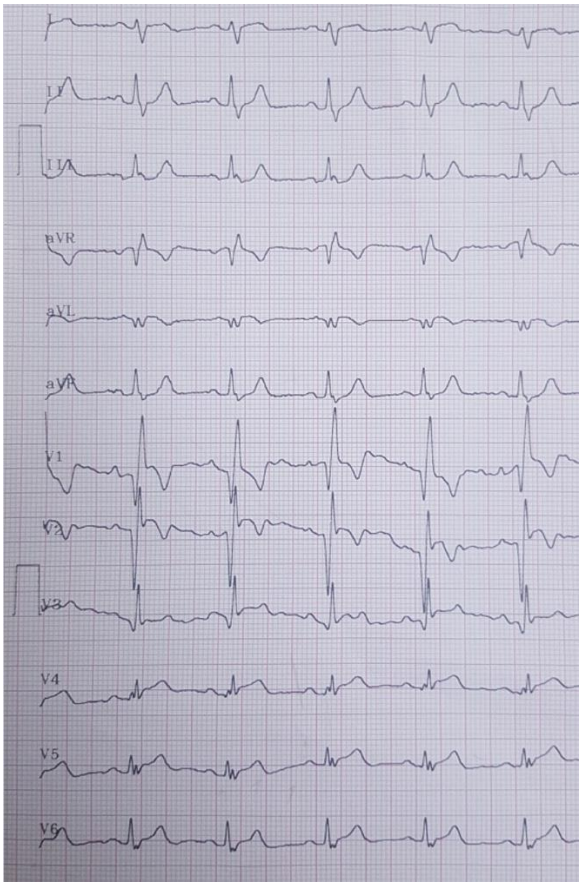

Figure 1. Electrocardiography

coronary intervention (PCI) at the hospital, the patient was immediately transferred to the coronary care unit (CCU) after being diagnosed with STEMI by the attending physician. Before being transferred to the CCU, the patient was administered supplemental oxygen via nasal cannula, statin, dual antiplatelet, and nitrates in the emergency department. The echocardiography performed in the CCU at the bedside revealed akinesia in the anterior circulation segments with an ejection fraction of approximately 15%. Ten units of Reteplase were administered and then repeated 30 minutes after the patient's arrival in the CCU. Positive cardiac markers were reported, and a complete blood count revealed leukocytosis. Blood pressure was approximately 100/55 mmHg, and basilar rales were still present. Multiple episodes of non-sustained ventricular tachycardia and accelerated idioventricular rhythm led to a partial resolution of his chest pain and ST-segment elevation.

The patient was referred to a PCI-capable center for coronary angiography, shown in clips 1-4. The patient had ectasia of the proximal left anterior descending (LAD) artery with a slow flow pattern, a thrombus in the same area, a severed diagonal branch, and a significant lesion between the two ectatic proximal and mid portions of the right coronary artery (RCA). Considering the lesions and the thrombus burden, it was decided that the patient would receive a dual antiplatelet and anticoagulant during admission and novel oral anticoagulants (NOACs) after discharge, with no intervention on the lesions.

Outcome and Follow-up

The patient was discharged in good health without chest pain while on dual antiplatelet, statin, and rivaroxaban treatment. Furthermore, the patient was administered a beta-blocker, an angiotensin-converting enzyme inhibitor, and a mineralocorticoid receptor antagonist. Twenty days after the event, the individual returned to the primary care center with exertional chest pain. ECG exhibited no new changes. Echocardiography was performed on the patient, as shown in clips 5-7. The patient had an improved ejection fraction of approximately 25% with significant wall motion abnormalities in the anterior segments. The treatment dosage was increased, and nitrates were added to the regimen. The patient reports normal health approximately 28 days after the incident.

Discussion

According to our knowledge, this is the first documented instance of a possible STEMI with ectatic coronary arteries following CO poisoning. CO binds to hemoglobin with a greater affinity than 200 times that of oxygen (O_2); therefore, even small amounts of CO reduce O_2 delivery to tissues such as the brain and heart, which are more sensitive to hypoxia than other tissues. Hypoxia impairs cellular oxygen consumption and the mitochondrial electron transport chain, leading to lipid peroxidation and free radical production. In addition, hypoxia promotes the production of hypoxic inducible factor 1 (HIF-1α) and, consequently, inflammation [8]. These effects may contribute to the myocardial damage observed in over a third of patients with CO poisoning [2]. In addition, CO and other air pollutants, including specific substances, cause a prothrombotic state, which increases the risk of ischemic heart disease and MI even under normal coronary conditions [8]. By increasing cardiac markers or ST-elevations on the ECG [3], transient ischemia can imitate the clinical course of a MI. In addition, despite a normal angiogram, the effect of CO on the mitochondrial energy consumption of myocytes can cause local hypokinesis of the ventricular walls, also known as a stunned-myocardium-like syndrome [9].

Although myocardial ischemia due to hypoxia and myopericarditis could have been the leading differential diagnosis in our case, the patient was treated for STEMI based on his symptoms and the physician's decision. The individual exhibited a thrombotic lesion on coronary angiography, possibly due to the thrombolytic effect on an occluded coronary or simultaneous thrombus formation in a patient with CO-

induced myocardial ischemia. The patient was successfully treated with thrombolytic and anticoagulant medications. Other case reports have also confirmed improved outcomes of treatment with percutaneous intervention (PCI) in CO-induced STEMI patients; however, in contrast to the current paper, the patients described in these reports had risk factors for ischemic heart disease that call into question the causality of CO poisoning [3-5].

Satran et al. retrospectively reviewed data from 230 CO poisoning patients and revealed that the male gender, Glasgow coma scale (GCS) score of 14, and hypertension are predictors of myocardial injury. Intriguingly, active cigarette smoking (CS) was significantly associated with a lower risk of myocardial damage [2]. A possible explanation for this phenomenon can be the long-term compensatory mechanisms adopted by myocytes to combat the Hypoxia-induced by accumulated CO when smoking cigarettes in confined spaces [8]. This is consistent with previous research confirming that smoking is associated with a lower risk of IHD-related mortality, thus demonstrating the smoker's paradox [10].

The growing prevalence of smokeless tobacco use in the Middle East has raised healthcare system concerns. Over 365 million people worldwide use smokeless tobacco, primarily young adults [11]. In addition, a study of over 50,000 adults in Iran revealed that 7% of the adult population uses smokeless tobacco. Although the risk ratio of smokeless tobacco for IHD-related mortality has increased, it has not reached a significant level [12]. Furthermore, tobacco use is the most preventable cause of cardiovascular diseases (CVD) [7]. Through multiple mechanisms, including endothelial dysfunction, oxidative stress, inflammation, and coagulopathy [13], smokeless tobacco can either accelerate or induce the occurrence of MI.

Given that our patient was abusing Nas, reports on the association of smokeless tobacco with CHD are contradictory [14]. Some studies proposed smokeless tobacco as a risk factor for acute MI or CHD [15,16]. Conversely, several studies failed to observe such an association and instead found a link between smokeless tobacco and fatal CHD [7,17-20]. Another study found a geographical difference in the incidence of CHD among users of smokeless tobacco. According to Aishwarya et al., smokeless tobacco significantly increased the incidence of non-fatal CHD in Asia but only for fatal CHD in Europe and the US [6]. Multiple factors, including smokeless tobacco dose, duration and frequency of consumption, type of processing and composing elements, and other traditional CVD risk factors, may be responsible for the disparity in geographical results [16]. These findings caution healthcare systems to implement strategies and policies to increase public awareness of the adverse effects of smokeless tobacco use. In conclusion, it appears that CO poisoning and smokeless tobacco consumption may have contributed to MI in our case.

Conclusion

We presented a case of MI in a young user of smokeless tobacco following CO poisoning and successful thrombolytic therapy. In patients with carbon monoxide poisoning, we recommend clinicians consider coronary heart disease's probability and obtain a baseline ECG and cardiac markers. We suggest that future research investigate these associations and the underlying mechanisms underlying this phenomenon.

Acknowledgment

None.

Conflict of interest

None.

Author Contribution

MMAR, NV, FH: manuscript review, manuscript editing. MMAR, NV, SM: manuscript preparation. MMAR, SM, FH: design and concepts. NV, MG, FH: literature search.

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