Prikaz dveh primerov zastrupitve z ogljikovim monoksidom

Carbon monoxide intoxication: A report of two cases

Avtor / Author

Ustanova / Institute

Emina Hajdinjak[1], Andrej Markota[2], Alenka Strdin Košir[2], Simona Kirbiš[2,3]

[1]Center za nujno medicinsko pomoč Maribor, Zdravstveni dom dr. Adolfa Drolca Maribor, Maribor, Slovenija; [2]Univerzitetni klinični center Maribor, Oddelek za intenzivno interno medicino, Maribor, Slovenija; [3]Univerza v Mariboru, Medicinska fakulteta, Maribor, Slovenija

[1]Centre for Emergency Medicine Maribor, Maribor, Slovenia; [2]University Medical Centre Maribor, Medical Intensive Care Unit, Maribor, Slovenia; [3]University of Maribor, Faculty of Medicine, Maribor, Slovenia

Ključne besede:
zastrupitev z ogljikovim monoksidom, naprave za monitoriranje bolnika, motnje zavesti.

Key words:
carbon monoxide intoxication, monitoring devices, unconsciousness.

Članek prispel / Received
10.08.2012
Članek sprejet / Accepted
29.10.2013

Naslov za dopisovanje / Correspondence
Andrej Markota, Univerzitetni klinični center Maribor, Oddelek za intenzivno interno medicino, Ljubljanska 5, SI–2000 Maribor, Slovenija
Telefon +386 23212471
E–pošta: andrejmarkota@hotmail.com

Izvleček

Namen: *Namen prispevka je na primeru dveh bolnikov prikazati uporabo neinvazivnih merilcev ogljikovega monoksida. Znaki zastrupitve z ogljikovim monoksidom so nespecifični; nanjo ponavadi pomislimo ob anamnestičnih podatkih in po izključitvi ostalih vzrokov za nezavest.*

Prikaz primerov: *V prispevku sta opisana primera dveh bolnikov, ki sta bila zastrupljena z ogljikovim monoksidom in bila akutno alkoholno opojena. Iz dostopnih podatkov o bolnikih (najdena na odprtem, nezavestna, brez zunanjih znakov za poškodbo) na tovrstno zastrupitev ni bilo mogoče sklepati; v obeh primerih smo zastrupitev z ogljikovim monoksidom odkrili s pomočjo neinvazivnega merilca ogljikovega monoksida, ki je bil uporabljen v sklopu ABCDE pristopa. Ostale meritve (krvni tlak, krvni sladkor ipd.) niso*

Abstract

Purpose: *Here, we describe the application of non–invasive carbon monoxide monitoring for two patients with suspected carbon monoxide intoxication.*

Case reports: *Since signs and symptoms are non–specific, carbon monoxide intoxication is often diagnosed only after other causes of unconsciousness have been excluded. This paper presents two cases of carbon monoxide and alcohol intoxication. From the information available (both patients were found in open spaces, unconscious, with no external signs of injury), it was impossible to infer carbon monoxide intoxication. In both cases, carbon monoxide intoxication was detected by noninvasive carbon monoxide monitoring in accordance with the ABCDE approach for the immediate assess-*

pojasnile stanja bolnikov. Bolnika sta prejela zdravljenje s kisikom še pred določitvijo ogljikovega monoksida v arterijski krvi in bila po dveh dneh zdravljenja odpuščena brez posledic. Vzroki za zastrupitev z ogljikovim monoksidom so bili izpostavljenost dimu in kajenje ob hkratni zastrupitvi z alkoholom.

Zaključek: *Uporaba enostavnih, neinvazivnih sistemov za merjenje ogljikovega monoksida lahko olajša postavitev diagnoze zastrupitev z ogljikovim monoksidom.*

ment and treatment of critically ill patients. Other findings (blood pressure, blood sugar, etc.) did not explain the state of either patient. Both patients received oxygen therapy before the determination of carbon monoxide in the arterial blood and both were discharged after 2 days of treatment without short–term neurological sequelae. The source of carbon monoxide in each case was exposure to smoke and automobile exhaust, respectively, with concurrent alcohol intoxication.

Conclusion: *The use of devices that enable the rapid and noninvasive detection of carbon monoxide can facilitate diagnosis of carbon monoxide intoxication.*

INTRODUCTION

Clinical signs of carbon monoxide (CO) intoxication are non–specific, especially in case of mild poisoning, where headache, dizziness and myalgia are present. In severe poisoning, which can be fatal, the patient becomes confused and disoriented, and may lose consciousness (1). CO intoxication is often unintentional with an incidence of about 16 cases per 100,000 persons in the US, resulting in about 450 deaths annually (2), and 2.4 cases per 100,000 persons in Slovenia (3). The difference in the incidence of CO intoxication between the US and Slovenia is most likely due to unrecognized mild poisoning cases. Unintentional CO intoxication is usually diagnosed only after other causes of unconsciousness have been excluded (1,3,4). CO toxicity is a result of a number of pathways. The affinity of CO for hemoglobin is about 200 times that of oxygen. Even with low amounts of inhaled CO, carboxyhemoglobin is formed, resulting in hypoxia. Additionally, independent of hypoxia, reactive oxygen species production and intracellular concentration of heme are increased, thereby interrupting cellular respiration and provoking a stress response. Subsequent cellular damage may lead to necrosis and apoptosis, especially of the neurons (1,2,4).

CO intoxication is treated by administration of 100% oxygen via non–rebreather masks or endotracheal intubation to unconscious patients. Treatment should be started as soon as possible and provided until carboxyhemoglobin levels fall to below 5%. Oxygen administration accelerates CO elimination and reduces oxidative stress and inflammation. These effects are more pronounced at high arterial and tissue oxygen tensions, as the basis for hyperbaric oxygen treatment (1,4).

CO intoxication is confirmed by blood gas analysis, which is only possible in a hospital environment. Non–invasive determination of CO based on pulse spectrometry allows immediate determination of CO saturation of Hb and immediate appropriate action in the prehospital environment or in an emergency room setting. Determination of CO saturation can have a significant impact on treatment outcome (5).

Here, we present two separate cases of 51– and 47–year–old males who were found unconscious in open spaces. During initial examinations in the prehospital and emergency room settings, a CO monitor revealed carboxyhemoglobin levels of 28% and 22%, respectfully.

CASE PRESENTATIONS

An unreponsive 51–year–old man was discovered along a roadway in an open suburban area by a coincidental passerby. The emergency medical unit (EMU) was contacted when the patient failed to respond

to voice. Upon arrival of the EMU, the patient was unconscious with an estimated Glasgow coma score (GCS) of 3 points. His breathing rate was 20 breaths/min, peripheral arterial blood oxygen saturation was 89% with a blood pressure of 100/60 mmHg, heart rate of 85 beats/min, blood glucose concentration of 5.4 mmol/L, and a body temperature of 35.8°C. There were no external signs of injury; however, the patient had a strong odor of alcohol and tobacco. Oxygen saturation improved immediately upon oxygen administration using an Ohio mask and the other vital functions remained unchanged. The EMU was equipped with a peripheral oxygen saturation monitor, which also automatically measures CO. Once the patient was attached to the monitor, his initial carboxyhemoglobin saturation was 28%. The patient was transported to a local hospital and during transport, the carboxyhemoglobin levels gradual decreased, reaching 10% just before patient hand–over. The time for prehospital treatment and transport was 37 min.

On arrival at the hospital, the patient remained unconscious with an estimated GCS of 3 and a peripheral oxygen saturation of 97% while continuing to use an Ohio mask. In addition to the basic biochemical tests and blood counts, ethanol and CO concentrations were determined, which revealed elevated levels of CO (8.3%) and ethanol (2.9 g/L) with no major discrepancies in other findings. Oxygen therapy was continued for 10 h until the patient attained full orientation (GCS 15). Controlled laboratory tests revealed a CO level of 2.0% and an ethanol concentration of 0.5 g/L with other findings within normal limits. The patient revealed that he was at a party and consumed alcoholic beverages while smoking in a small, enclosed space with five other smokers. Additionally, they were exposed to an open fire burning in a fireplace. He did not remember walking outside. Other participants denied any health problems. After 26 h of treatment he was discharged to home care.

The second patient was a 47–year–old man. Passers–by contacted EMT because of inebriation. Upon arrival of the EMT, his GCS was estimated at 10 points. His peripheral arterial blood oxygen saturation was 94%, blood pressure 130/60 mmHg, and heart rate 95 beats/min. His blood glucose concentration was 7.4 mmol/L. There were no external signs of injury; however, the patient had a strong odor of alcohol. An intravenous line was inserted for saline infusion and the patient was transported to a local hospital. Prehospital treatment and transport lasted 45 min.

On arrival at the hospital emergency department, his GCS was estimated at 11–12, blood pressure was 136/72 mmHg, peripheral arterial blood oxygen saturation was 96%, and heart rate was 95 beats/min. The peripheral arterial oxygen saturation monitor was equipped with a CO monitor, which automatically alerts the operator to increased CO levels. The monitor revealed CO levels of 22%. In addition to blood tests to determine basic biochemical tests and blood counts, ethanol and CO concentrations were determined to be 18% and 3.3 g/L, respectively, with no major discrepancies in other findings. The patient's condition improved with oxygen administration with an Ohio mask and fluid infusion. After 12 h of treatment, he regained full consciousness with an estimated GCS of 15 points. Control tests revealed CO levels of 5.6% and ethanol of 1.0 g/L with other findings within normal limits. The patient subsequently revealed that he spent about 5 h in a garage repairing a car, with the engine periodically running. Additionally, he was smoking and drinking alcohol. He remembered becoming nauseous, but had no memory of later events. After 36 h of in–hospital treatment, he was discharged to home care.

DISCUSSION

CO intoxication is difficult to diagnose because of the lack of specific signs and symptoms, and a history of CO exposure is often unavailable at presentation (1–4). Therefore, a definitive diagnosis is confirmed by determining blood carboxyhemoglobin levels (1,4). Carboxyhemoglobin levels can also be determined using non–invasive analyzers; however, such devices are not yet widely available (5,6). Nonetheless, non–invasive analyzers are portable, easy to use, and the measurement results can influence the course of treat-

ment (5,6). Point–of–care devices enable rapid implementation of treatment with 100% oxygen, which is safe, available in both prehospital and hospital environments, and inexpensive (1). Early recognition of high–risk patients using point–of–care devices could help the clinician to determine which patients would benefit from hyperbaric oxygen treatment (1). The use of portable, non–invasive carboxyhemoglobin analyzers is possible in prehospital, emergency room, and intensive care unit settings. The diagnosis was delayed in the two presented cases because of concurrent alcohol intoxication, which is an easily recognizable factor influencing the state of consciousness.

In the first case, the EMT team that responded to the call had a LIFEPAK®15 (PhysioControl™) monitor, which is equipped with a peripheral arterial oxygen saturation monitor (Masimo Rainbow SET® DCI–DC12). In the second case, the EMT team used a hand–held Masimo Rainbow SET® Rad–57 peripheral arterial oxygen saturation monitor. Both monitors automatically measure oxygen, CO, and methemoglobin saturation. CO detection is based on absorption of light at different wavelengths corresponding to peak absorption values for Hb, oxygenated Hb, carboxyhemoglobin, and methemoglobin. The results regarding carboxyhemoglobin saturation, taking into account the limitations imposed by the method itself, were comparable to determinations of carboxyhemoglobin in the blood (6). In case of hypotension and hypoperfusion of the extremities, these measurements are not reliable. The same limitations apply to the widely used peripheral arterial oxygen saturation monitors (6).

CO determination in an emergency setting can also be performed using devices that measure CO in exhaled air. Such devices are simple to use and portable with obtained CO measurements that are comparable with those from arterial blood samples. The percentage of exhaled CO is determined by changes in the electrical potential of the electrode due to CO oxidation (7).

Smokers have relatively higher carboxyhemoglobin levels because of CO inhalation via cigarette smoke. Although both of our patients were smokers, there were no signs of acute CO intoxication Additional sources of CO were smoke from a fireplace in the first case and automobile exhaust in the second. In the first case, other party attendees reported no abnormal body functions; however, carboxyhemoglobin levels were not tested. In addition to smokers, increased CO saturation may also present in those who are chronically exposed to a source of CO either at home or work due to gas heaters, open ovens, car exhaust, highway workers, and exposure to incomplete hydrocarbon combustion. In such patients, carboxyhemoglobin levels can reach 8–10% without exhibiting signs of acute intoxication (8). In some patients, the presence of CO in the blood can be an accidental finding and not the cause of the patient's condition. In our case, both patients were also positive for acute alcohol intoxication. For both cases, we administered oxygen therapy (via Ohio mask) and fluid infusion. For the first patient, we decided against intubation even though he was deeply unconscious (GCS 3 on admission) because his state of consciousness rapidly improved.

Patients with CO poisoning should be followed after discharge because it is difficult to predict the extent and rate of recovery. Long–term management can be associated with complicated sequelae, which can develop weeks after poisoning and become permanent. Unfortunately, there is no established therapy for sequelae after CO poisoning (1,2). For the two presented cases, follow–up was arranged via the patients' family doctors.

CONCLUSION

Clinical signs of CO intoxication are non–specific. Often, only an appropriate history with a possible source of CO points to CO intoxication. The diagnosis is often one of exclusion. Our cases demonstrate the effective use of a simple method that can be performed in a prehospital or emergency room setting, which can significantly affect the course of treatment. The use of devices that enable rapid and noninvasive patient monitoring can facilitate diagnosis of CO intoxication.

REFERENCES

1. Weaver LK. Clinical practice. Carbon monoxide poisoning. N Engl J Med 2009; 360(12): 1217–25.
2. Hampson NB, Piantadosi CA, Thom SR, Weaver LK. Practice recommendations in the diagnosis, management, and prevention of carbon monoxide poisoning. Am J Respir Crit Care Med 2012; 186(11): 1095–101.
3. Brvar M , Jamšek M , Možina M, Horvat M, Gorjup V. Epidemiološki pregled zastrupitev z ogljikovim monoksidom v Ljubljani od 1990 do 1999. Zdrav Vest 2002; 71: 87–90.
4. Centers for Disease Control and Prevention (CDC). Nonfatal, unintentional, nonfire–related carbon monoxide exposures in the United States, 2004–2006. Morb Mortal Wkly Rep 2008; 57(33): 896–9.
5. Nilson D, Partridge R, Suner S, Jay G. Non–invasive carboxyhemoglobin monitoring: screening emergency medical services patients for carbon monoxide exposure. Prehosp Disaster Med 2010; 25(3): 253–6.
6. Barker SJ, Curry J, Redford D, Morgan S. Measurement of carboxyhemoglobin and methemoglobin by pulse oximetry: a human volunteer study. Anesthesiology 2006; 105(5): 892–7.
7. Andersson MF, Moller AM. Assessment of carbon monoxide values in smokers: a comparison of carbon monoxide in expired air and carboxyhaemoglobin in arterial blood. Eur J Anaesthesiol 2010; 27(9): 812–8.
8. Sato K, Tamaki K, Hattori H, Moore CM, Tsutsumi H, Okajima H et al. Determination of total hemoglobin in forensic blood samples with special reference to carboxyhemoglobin analysis. Forensic Sci Int 1990; 48: 89–96.