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-
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. 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.

Case Presentations

An unresponsive 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 gradually 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 imple-
mentation of treatment with 100% oxygen, which is
safe, available in both prehospital and hospital envi-
ronments, 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 analy-
zers is possible in prehospital, emergency room, and
intensive care unit settings. The diagnosis was delayed
in the two presented cases because of concurrent alco-
hol 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 peri-
pheral arterial oxygen saturation monitor. Both moni-
tors automatically measure oxygen, CO, and meth-
emoglobin saturation. CO detection is based on ab-
sorption of light at different wavelengths correspon-
ding 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 hypo-
tension 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 lev-
els because of CO inhalation via cigarette smoke. Al-
though 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 satu-
ration may also present in those who are chronically ex-
posed 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 conscious-
ness 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 de-
velop weeks after poisoning and become permanent.
Unfortunately, there is no established therapy for
sequelae after CO poisoning (1,2). For the two pre-
sented 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 diag-
nosis is often one of exclusion. Our cases demon-
strate 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 diag-
nosis of CO intoxication.
REFERENCES