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Carbon monoxide poisoning-induced upper gastrointestinal bleeding

Introduction

Carbon monoxide (CO) is a colorless, odorless, tasteless, and non-irritating gas that is due to the incomplete combustion of carbon-borne fuels [1]. Cases of CO poisoning are common in Turkey because of social, economic, and climatic reasons. Carbon monoxide is also an important agent in the world of poisoning-related deaths [2, 3]. Poisoning symptoms are predominant in the cardiovascular, respiratory, and neuropsychiatric systems, whereas the genitourinary and gastrointestinal systems are rarely affected [4]. Here, we report the case of a patient with upper gastrointestinal bleeding after CO poisoning, which has not been published before in the literature.

Case presentation

A 61-year-old female patient was seen unconscious in her room by her relatives at 12:10 p.m. She was admitted to our emergency department with 0.9% NaCl fluid delivered via a peripheral intravascular cannula, and oxygen was given with a flow rate of 4 l/min through nasal prongs during the transportation time (approximately 25 min) by the Emergency Medical Services at 12:45 p.m. Her Glasgow coma scale (GCS) score was 5, and the vital signs were as follows: blood pressure: 60/40 mm Hg; pulse rate: 140/min; respiratory rate: 24/min; and body temperature: 36.2°C. Physical examination for bilateral Babinski reflexes revealed downward response of the hallux, and the pupils were isochoric. Except for hypertension and panic disorder, there was

not any chronic disease in the patient's medical history. The patient was intubated with 8 mg midazolam and 50 mg rocuronium owing to her GCS score, and 100% oxygen support was provided by a mechanical ventilator. After intubation, she had bright red vomiting. We inserted a nasogastric tube to determine the cause of the upper gastrointestinal tract bleeding. There was a 400 cc bloody nasogastric aspirate, which confirmed the diagnosis of upper gastrointestinal hemorrhage. Subsequently, 0.9% NaCl and hydroxyethyl starch fluids were used to replace the plasma volume for the hemodynamically unstable patient. An intravenous 80 mg esomeprazole bolus injection was used to decrease the gastric acidity.

The patient had no history of upper gastrointestinal bleeding, ulcers, anticoagulant use, hepatitis, cirrhosis, and malignancy. Her relatives reported a slight smell of smoke in the patient's bedroom. Arterial blood gas results after endotracheal intubation management (12:50 p.m.) were as follows: pH: 7.22; pCO₂: 27.5 mm Hg; pO₂: 86.5 mm Hg; SaO₂: 97.6%; HCO₃⁻: 10.8 mEq/l; carboxyhemoglobin (COHb): 33.6%; Anion Gap: 28.2 mmol/l; lactate: 13.6 mmol/l. In the complete blood count the following parameters were measured: white blood cells: 27,210/ul; platelets: 476,000/uL; Hgb: 9.1 g/dL; aPTT: 23.6 sec; prothrombin time was measured as 12.2 sec. Magnetic resonance imaging of the brain and the clinical results excluded cerebrovascular diseases. Finally, the patient was hospitalized in the intensive care unit with

the diagnosis of upper gastrointestinal bleeding after CO poisoning. The patient was managed symptomatically, and a three-unit erythrocyte suspension was infused. Arterial blood gas results 4 h after hospitalization in the intensive care unit were as follows: pH: 7.45; PaO₂: 64.9 mm Hg; pCO₂: 24.8 mm Hg; SaO₂: 93.3%; HCO₃⁻: 17.1 mEq/l; COHb: 3.7%; lactate: 4.3 mmol/l. The patient was extubated after 8 h, and on the second day, the GCS score was 15. Endoscopic examination revealed multiple bleeding sites as pan-erosive gastritis. The patient was discharged on the fifth day without any complications.

Discussion

Carbon monoxide has a 210-fold greater affinity than oxygen to bind to hemoglobin. It forms COHb, which reduces the amount and release of oxygen to the tissues. As a result, CO leads to relative anemia, causing asphyxia or tissue hypoxia [5]. As the stage of CO toxicity increases, cytochrome aa₃ is inactivated, and disruption of cellular respiration begins [6].

Clinical manifestations of CO intoxication are quite extensive. Clinical symptoms are nonspecific. Headache is the first symptom, and multiple systems can be affected by the poisoning severity, which can be fatal [7]. While COHb induces rhabdomyolysis by decreasing oxygen supply to the muscle tissues [8], CO is associated with leukocyte-dependent inflammatory changes and lipid peroxidation in the brain. Demyelination in the white matter, especially edema and

focal necrosis in the globus pallidus, are also seen [3].

The frequency of gastrointestinal bleeding is 103/100,000 per year, and more than 75% of all gastrointestinal bleeding originates from the upper gastrointestinal part. The mortality rate in acute upper gastrointestinal hemorrhage is estimated at 2–10% despite intensive care [9].

After CO exposure, gastrointestinal symptoms such as nausea and dyspepsia are common, but gastric ulcer and hemorrhage are rare [10]. Bleeding because of CO intoxication is thought to be due to mucosal lesions caused by hypoxia and stress. Although the pathogenesis of acute gastric mucosal lesions induced by hypoxia is not definitively understood, Kamiyama et al. determined that cold and hypoxic stress induce acute gastric mucosal lesions. Kamiyama et al. have found a correlation between the decrease of potential difference in the gastric mucosa and the gastric mucosa defensive mechanism [11, 12]. Naito et al. reported that the peroxidation of phosphatidylcholine levels among patients at high altitudes was higher when compared with their control group, which was accepted as a risk factor for gastrointestinal bleeding [13]. In a study conducted by Walther on rabbits, mucosal erosion, hemorrhage, and edema were determined in the microscopic examination of a gastric biopsy taken after acute CO poisoning [14].

Conclusion

Although gastrointestinal complications are rare after CO poisoning, it should be kept in mind that gastrointestinal bleeding may develop owing to hypoxia-induced gastric mucosal lesions and preventive treatment should be provided.

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Compliance with ethical guidelines

Conflict of interest Y.K. Tekin, İ. Korkmaz, E. Demirtaş, B. Günday, and G. Tekin declare that they have no competing interests.

All procedures performed in studies involving human participants or on human tissue were in accordance with the ethical standards of the institutional and/or national research committee and with the 1975 Helsinki declaration and its later amendments or comparable ethical standards. Informed consent was obtained from all individual participants included in the study.

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