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Case Report [Olgu Sunumu]

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A very rare complication of carbon monoxide intoxication; thrombotic thrombocytopenic purpura

[Karbonmonoksit zehirlenmesinde nadir bir komplikasyon: Trombotik trombositopenik purpura]

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ABSTRACT

Carbon monoxide intoxication is an important public health problem, which may result in death. A 46-year-old male patient was found in an unconscious state at home and was transported to the emergency service by the patient's relatives. Relatives of the patients were indicated that there was gas leak from the stove. At first it was considered that the patient might be poisoned with carbon monoxide. The laboratory findings were, creatinine 2.5 mg/ dL and creatinine kinase 88.15 U/L. Carboxy hemoglobin level was 30.9%. Hyperbaric oxygen therapy was commenced with the diagnosis of carbon monoxide intoxication. The laboratory values in the 2nd day of admission were, creatinine 5.8 mg/ dL, creatinine kinase 88.42 U/L. Then the patient underwent a hemodialysis. Hemoglobin levels decreased to 7.3 g/dL, platelet count dropped to 11.000/mm^3. Peripheral blood smear showed erythrocyte fragmentation. Thrombotic thrombocytopenic purpura was considered and methylprednisolone and plasmapheresis was started. After the 10th session of plasmapheresis, platelet value was over 150.000/mm^3 and plasmapheresis was discontinued. Platelet value was 285.000/mm^3 and creatinine 0.7 mg/dL. The patient was discharged. Thrombotic thrombocytopenic purpura which might have complication should be kept in mind in patients with carbon monoxide intoxication.

Key Words: Carbon monoxide intoxication, thrombocytopenic purpura
Conflict of Interest: The authors declare no conflict of interest.

ÖZET

Karbonmonoksid zehirlenmesi ölümle sonuçlanabilen önemli bir halk sağlığı sorunudur. Evinde bilinç kaybı gelişmiş bir şekilde bulunan 46 yaşında erkek hasta acil servise getirildi. Hasta yakınları hastayı evde bulduklarında sobadan gaz sızıntısı olduğunu ifade ettiler. Bunun üzerine hastada karbonmonoksid zehirlenmesi gelişmiş olabileceği düşünüldü. Hastanın yapılan laboratuvar testlerinde kreatinin 2,5 mg/dL, kreatin kinaz 88.15 U/L olarak bulundu. Karboksihemoglobin düzeyi %30.9 idi. Hastaya CO zehirlenmesine yönelik hiperbarik oksijen tedavisi başlandı. Yatışının 2. gününde kreatinin 5.8 mg/dL, kreatin kinaz 88.42 U/L olarak bulundu. Hasta hemodiyaliz tedavisine alındı. Hastanın hemoglobin düzeyi 7.3 g/dL'e, trombosit sayısı 11.000/mm^3'e düştü. Yapılan periferik yaymada parçalanmış eritrositlerin olduğu görüldü. Hastada trombotik trombositopenik purpura gelişmiş olabileceği düşünülerek metilprednizolon ve plazmaferez tedavisine başlandı. 10 seans plazmaferez tedavisinden sonra hastanın trombosit değeri 150.000/mm^3 üzerinde seyrettiği görülerek plazmaferez tedavisi sonlandırıldı. Hastanın trombosit değerinin 285.000/mm^3, kreatinin düzeyinin 0.7 mg/dL olması üzerine hasta taburcu edildi. Trombotik trombositopenik purpura, karbon monoksit zehirlenmesi olan hastalarda bir komplikasyon olarak akılda tutulmalıdır.

Anahtar Kelimeler: Karbonmonoksit zehirlenmesi, trombositopenik purpura
Çıkar Çatışması: Yazarların çıkar çatışması yoktur.

Introduction

Carbon monoxide (CO) intoxication is an important public health problem that should not be ignored. CO is a highly toxic gas and easily absorbed through the lungs. It can cause poisoning [1]. Most intoxications are caused by exhaust gases from coal stoves and gasoline and by the occurrence of symptoms in relation to a possible exposure. Early diagnosis and treatment reduce the morbidity and mortality rates [2]. Thrombotic thrombocytopenic purpura (TTP) is a rare condition, characterized by fever, acute renal failure, thrombocytopenia, microangiopathic hemolytic anemia (MAHA), and neurological abnormalities [3]. In this study we report a case of carbon monoxide intoxication complicated with TTP in a 46 year old male patient.

Subject and Methods

A 46-year-old male patient was found in an unconscious state in home by his relatives and he was brought to the emergency service. His vital signs, respiratory, abdominal and cardiovascular system findings were normal. Although the patient was conscious, he had a tendency to sleep. In neurological examination, there was loss of strength and hypoesthesia in the right upper extremity. His relatives stated that there was gas leak from the stove in the home when he was found. As a presumable diagnosis, CO intoxication was considered. The laboratory findings were as follows, hemoglobin 16.1 g/dL, platelet count 261.000/mm^3, blood urea nitrogen (BUN) 56 mg/dL, creatinine 2.5 mg/dL, Na 146 mmol/L, K 4.1 mmol/L, lactate dehydrogenase (LDH) 1472 U/L and creatinine kinase (CK) 88.15 U/L. In arterial blood gas analysis performed upon admission, PO_2 was 75 mmHg; PCO_2 was 30 mmHg and pH was 7.48. Carboxyhemoglobin (COHb) level was 30.9%. ECG recordings and chest x-ray were normal. In cranial computed tomography, there was a hypodense area with contours on the lobule and rising to the vicinity of left lateral ventricle frontal horn. With the diagnosis of cerebrovascular event and simultaneous CO intoxication, low molecular weight heparin and hyperbaric oxygen treatment was commenced. The laboratory values in the 2nd day of admission were as follows, BUN 235 mg/dL, creatinine 5.8 mg/dL, K 6.6 mmol/L, phosphorus 8.2 mg/dL and CK 88.42 U/L. Urinary output was decreased. There was no bacterial growth in the urine culture. Abdominal ultrasound was normal. Tests for HBsAg, Anti HCV, p-ANCA and c-ANCA were negative. Then the patient underwent a daily hemodialysis program without heparin with the presumptive diagnosis of acute kidney injury due to rhabdomyolysis. Body temperature increased to 38.5º C at the same day, the level of consciousness appeared to be fluctuating, and there was a progressive decrease in platelet counts and hemoglobin. Hemoglobin levels decreased to 7.3 g/ dL, platelet count dropped to 11.000/mm^3 and LDH level was increased to 2212 U/L. INR, PT and aPTT values were normal.

Table 1. Results of the patient after plasmapheresis

	Results
Hemoglobin (g/dL)	9.8
Platelet (number per mm^3)	285.000
Creatinine (mg/dL)	0.7

Results

Peripheral blood smear showed erythrocyte fragmentation. TTP was considered and methylprednisolone and plasmapheresis was started. After the 1st session of plasmapheresis, the body temperature of the patient returned to normal and following the 4th session, the platelet count started to rise. After the 10th session of plasmapheresis, platelet values were over 150.000/mm^3 for two days and plasmapheresis was discontinued. Steroid dose was tapered to 4 mg a day and then withdrawn. The patient's general condition improved and urinary output increased. Patient's laboratory results after plasmapheresis were shown in Table 1. The patient was discharged with recommendation.

Discussion

CO intoxication is one of the most common types of poisoning across the world [2]. As the affinity of CO for Hb is 200-250 times higher than that of oxygen, it competitively inhibits the hemoglobin binding capacity of oxygen. It prevents O_2 transport and release of oxygen to the tissues. CO causes a relative anemia, leading to asphyxia and tissue hypoxia [4]. At the cellular level, damage is presumably due to a combination of hypoxia and a direct toxic effect of CO on mitochondrial function [1]. While the symptoms may be headache, fatigue and nausea in mild exposures, loss of consciousness, coma and even death may ensue in severe exposures [5].

Almost all organs are affected in CO intoxication especially organs that consume high levels oxygen such as brain, heart, and kidney. CO leads to a decrease in PO_2 in the muscle tissue and may cause rhabdomyolysis, which may result in kidney damage. CO may cause focal necrosis and edema in some brain regions, resulting in plegia and paresia in various organs and also changes can be seen in consciousness withpsychiatric symptoms [5]. In the case presented herein, on the 4th day of intoxication, TTP with the classical clinical pentad developed. There are reports on patients with carboxyhemoglobinemia who develop thrombocytopenia, however, the effect of CO on platelets is not well documented [6,7]. As far as we know, this is the second case of TTP accompanying CO intoxication [7]. Although the exact cause of TTP in CO intoxication remains unknown, the probable etiology may be associated with the effect of CO causing endothelium injury and its impact on platelets. Furthermore, the au-

thors of the first case report suggested that severe respiratory hypoxia may also lead to TTP [6]. However, in our patient respiratory hypoxia or circulatory failure was not seen when TTP developed. Exposure to CO results in focal intimal degeneration, cellular hypertrophy, splitting of subintimal structures, and endothelial edema in the aortas of mice [8]. In CO intoxication, CO and nitric oxide and its derivatives such as peroxynitrites disturb mitochondrial mechanisms and produce free oxygen radicals. Free oxygen radicals may give rise to vascular oxidative stress [9]. They may also have an impact on platelet aggregation and blood flow to contribute to endothelial damage and subsequent thrombosis intramurally. Moreover, free oxygen radicals inhibit endothelium derived relaxing factor and prostacyclin production by influencing vessel tone and platelet function, resulting in an increase in thrombus formation [10].

In conclusion, vascular damage that occurs in CO intoxication may trigger TTP in susceptible persons. In CO intoxication, it should be kept in mind that TTP may also develop due to CO and/or hypoxemia. Pathological process developing in the course of CO intoxication may trigger TTP, which may be fatal. In the monitoring of the patients diagnosed with CO intoxication, hematological parameters should be monitored.

Conflict of Interest

There are no conflicts of interest among the authors.

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