CASE REPORT

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Acute Lung Injury Due To Carbon Monoxide Exposure

ABSTRACT

A 20-year-old woman, who was found unconscious in the bed by the morning, was brought to emergency department. Her carboxyhemoglobin level was 20.2%. The portable chest X-ray showed bilaterally alveolar and interstitial infiltration. Initial pO_2/FIO_2 ratio was calculated as 119 mmHg. Acute lung injury due to carbon monoxide intoxication was considered. She was intubated and mechanical ventilation was applied. In the second day of hospitalization, a clear improvement was observed on the chest X-ray. She was discharged without any complication on the seventh day of hospitalization. Early diagnosis and treatment may prevent progression of ARDS and progression of permanent damage, and may lead to complete recovery.

Keywords: Carbon Monoxide Poisoning, Acute Lung Injury

Karbonmonoksit Zehirlenmesine Bağlı Ani Gelişen Akciğer Hasarı

ÖZET

Sabah yatağında bilinci kapalı olarak bulunan 20 yaşında bayan hasta Acil Servise getirildi. Karboksihemoglobin (COHb) değeri 20.2 idi. Çekilen portabl göğüs grafisinde her iki akciğerde alveolar ve intertisyel infiltrasyonlar görüldü. İlk pO_2 /FiO_2 oranı 119 mmHg ölçüldü. Karbon monoksit (CO) zehirlenmesine bağlı akut akciğer hasarı düşünüldü. Hasta entübe edildi ve mekanik ventilatöre bağlandı. Hastanın yatışının ikinci gününde çekilen akciğer grafisinde belirgin bir düzelme tespit edildi. Yatışının yedinci gününde hasta komplikasyonsuz olarak taburcu edildi. Erken tanı ve tedavi, akut solunum sıkıntısı sendromunun (ARDS) gelişmesini ve daha ileri hasar oluşumunu önleyebilir ve tam iyileşme ile sonuçlanabilir.

Anahtar Kelimeler: Karbonmonoksit Zehirlenmesi, Akut Akciğer Hasarı

INTRODUCTION

Carbon monoxide (CO), which occurs as a result of incomplete burning of carbon containing materials, is a colorless, odorless, tasteless, non-irritant and a fairly stable molecule that can be easily absorbed from the lungs (1). With its acute effects, CO intoxication can potentially be mortal and its delayed effects may lead to crucial consistent defects (2). High-oxygen-extracting organs, such as the brain, the heart, muscles and rarely lungs, easily become dysfunctional from CO intoxication (3). Due to CO exposure, lung diseases as pneumonia, pulmonary edema and respiratory distress have been reported so far (2). Here, we aim to present a case with CO intoxication resulted with acute lung injury.

CASE

A confused 20-year old woman was brought to emergency department (ED). From the patient history, it was found out that she had slept alone in a small room that was heated by a coal stove, and her family found her unconscious in the bed in the morning. Her previous medical history was unremarkable. Her Glasgow Coma Score (GCS) was 10 (E3M5V2) and she was agitated. She was tachypneic, tachycardic and central cyanosis was present. Pulse oximetry measured her oxygen saturation (SaO_2) as 45%. Auscultation of lungs revealed extensive bilateral pulmonary rales. Normobaric O2 therapy had been started at 10 L/min. The patient was intubated because of respiratory distress. Her initial blood pressure was 88/57 mmHg. Electrocardiography showed sinus tachycardia (120 beats/min) and 1mm ST depression on DII, DIII and aVF (Figure 1). Initial arterial blood gas (ABG) levels before oxygen therapy were as follows: pH: 7.09, pCO_2: 65.2 mmHg, pO_2: 24.9 mmHg, HCO_3: 20 mmol/L, BEecf: -10.0 mmol/L, BEb: -10.2 mmol/L, lactate: 4.2 mmol/L, SaO2: 43.8% and the carboxyhemoglobin (COHb) level was 20.2%. The pO2/FIO2 ratio and anion gap were calculated as 119 mmHg and 12, respectively. The hematologic, biochemical and toxicological parameters were normal. Cardiac biomarkers suggested myocardial ischemia [CK-MB: 48 U/L (normal range: < 5), Myoglobin: 56 µg/L (normal range: 10-46), Troponin I: 0.34 µg/L (normal range <0.1)]. Portable chest X-ray showed bilateral alveolar and interstitial infiltration (Figure 2a). Bedside echocardiography was normal (Ejection Fraction: 60%, global contractility, left ventricular chamber size, systolic function and wall motion: normal). The cranial computerized tomography demonstrated minimal edema possibly due to hypoxia. No typical lesions in cerebral white matter and basal ganglia were observed. As a result of all these findings, noncardiogenic pulmonary edema and ARDS due to CO intoxication were considered and she was sent to intensive care unit. Mechanical ventilator support with positive end expiratory pressure (PEEP) was provided to the patient. Hyperbaric oxygen treatment was unavailable in our region. After 24 hours, the patient's ABG values improved. Her pO_2/FIO_2 ratio was measured as 425.8 mmHg. In the second day of hospitalization, a clear improvement was observed on the chest x-ray (Figure 2b). She was weaned off from mechanic ventilator due to clinical improvement and then she was extubated. Troponin I peaked to 0.60 on the second day, and began to decrease afterwards. As her improvement was early, she was diagnosed as acute lung injury. Her blood cultures were negative and bronchoalveolar lavage showed normal flora. She was discharged without any complication on the seventh day of hospitalization. On the follow up after one month, no neuropsychological sequela was observed.

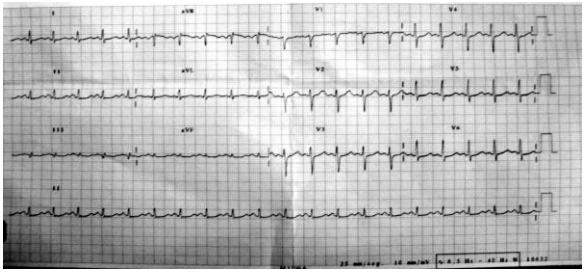

Figure 1. The patient's derived 12-lead ECG.

DISCUSSION

In chronic exposures, COHb levels do not correlate well with the clinic (4). In acute intoxicated patients with COHb levels of about 20%, the symptoms are relatively insignificant and consist of headache and dizziness (2). Contrary in chronic exposures, the same CO levels may result with more serious clinical presentations (4). Our patient was unconscious and cyanotic when she was admitted to ED with a CO level of 20%. This may be due to accompanying lung damage to acute exposure.

Lung findings related to CO exposure is rare (5). The lung damage seen in CO poisoning is probably related to impaired oxygen transport by the blood and is not a result of direct histotoxicity of the alveolar CO. The pulmonary changes in acute CO poisoning is attributed primarily to prolonged hypoxia (5). It is accepted that CO do not have direct toxic affect on lungs. But, animal studies showed that CO intoxication may lead to edema in endothelial cells and in interstitial space, and it may also damage alveolar Type II cells (6). This factor may affect capillary permeability and cause pulmonary edema. The most common clinical feature of respiratory system is pneumonia, and the second is pulmonary edema. Rarely adult respiratory distress syndrome (ARDS) develops following CO poisoning (2).

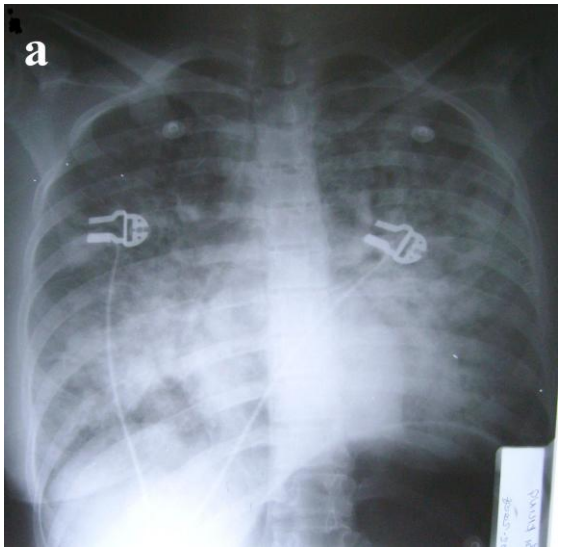

Figure 2a. The initial chest x-ray of the patient in the ED.

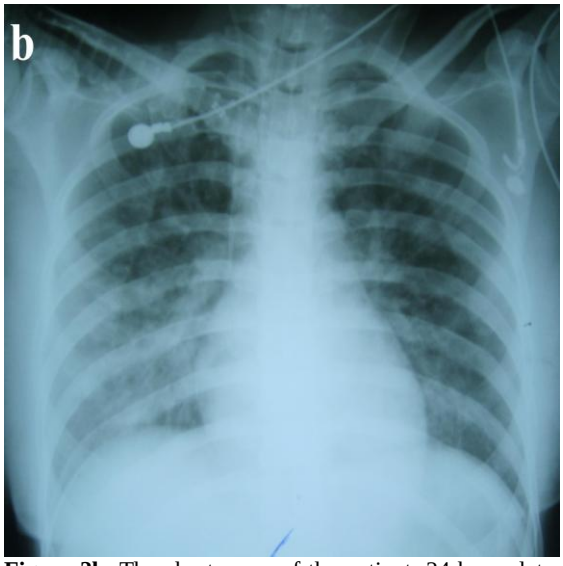

Figure 2b. The chest x-ray of the patient; 24 hours later after admittance to ED

In our case, the lung damage occurred after CO exposure, chest x-ray findings were compatible with lung edema, echocardiographic evaluation was normal, and our patients was young without no history of disease. All these revealed that the findings were due to CO intoxication.

Naeije et al. and Wu et al. described cardiogenic pulmonary edema due to CO exposure (7,8). But we observed noncardiogenic pulmonary edema in our case. Both cardiogenic and noncardiogenic pulmonary edema results from acute fluid accumulation in the alveoli, with resultant varying degrees of oxygen desaturation and respiratory distress. The reason of fluid accumulation in noncardiogenic pulmonary edema is generally due to permeability changes in the pulmonary capillary membrane itself (5). Fein et al. also showed that a patient who exposure to auto exhaust developed increased permeability-type pulmonary edema demonstrated by a normal capillary wedge pressure and production of protein-rich edema fluid. In their patient, there were many areas of denuded epithelium, hyaline membranes, and regenerating type II cells (9).

CO has got 250 times more affinity to Hb compared with O_2. This results in right shift of Hb-O_2 dissociation curve. Tissue hypoxia can firstly be seen in tissues that need O_2 more such as brain and heart. Cardiac damage due to COHb can be seen in even young adults or adolescents as in our case. Cardiac biomarkers may increase gradually. Myoglobin rises first in 2-4 hours, following CK-MB 4-6 hours. Troponin increases and decreases last, but it is most sensitive and specific for cardiac injury. For that reason, every patient with CO poisoning should be evaluated for cardiac injury in the ED (10).

Noncardiogenic pulmonary edema also is called ARDS (5) and the milder form of ARDS is termed as acute lung injury (ALI) (11,12). In both ARDS and ALI, pulmonary edema is seen in chest X-ray in a few hours after hypoxemia and this finding is followed by bilateral alveolar and interstitial infi1trates. But cardiomegaly and pleural effusion are not observed. These patients usually are in the need of mechanical ventilation (13). In our case, the patient had typical findings on chest X-ray. She had also respiratory acidosis and central cyanosis. Mechanic ventilator support was given immediately. Although the patients initial clinical examination and pO_2/FIO_2 ratio supported that the diagnosis may be ARDS, the patient's clinical and laboratory findings improved in 24 hours. Thus, we put the diagnosis as ALI to our patient.

In conclusion, CO intoxication may cause ALI and the patient may need early mechanical ventilator support. Early diagnosis and treatment may prevent progression of ARDS and progression of permanent damage, and may lead to complete recovery.

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