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A case report

Delayed encephalopathy after acute carbon monoxide poisoning:

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تأخر الاعتلال الدماغي الحاد بعد التسمم بأول أكسيد الكربون:

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الملخص العربي

الملخص: هذا تقرير عن حالة طفل عمره خمس سنوات يشتبه في تعرضه لتسمم حاد بأول أكسيد الكربون أحضره رجال الشرطة في حالة إغماء إلى غرفة الطوارئ. لوحظ تحسن كبير في الحالة العصبية للمريض بعد العلاج المبدئي بـ100% أكسجين باستخدام التهوية الميكانيكية. بعد ثلاثة أيام من التحسن، ظهر على المريض اضطراب عصبي حاد مفاجئ يشير إلى اعتلال دماغي متأخر. تمت معالجة المريض مرة أخرى باستخدام 100% أكسجين باستخدام التهوية الميكانيكية وعلاجات مساعدة أخرى. تمت إزالة الأنبوب من المريض في اليوم العاشر و قد خرج المريض من المستشفى بعد أسبوعين. بعد ستة أشهر من معالجة المريض بواسطة العلاج الطبيعي المكثف والتأهيل والمتابعة تم تقريبا علاج المريض.

الكلمات الدالة: التسمم بأول أكسيد الكربون، اعتلال الدماغ المتأخر

ABSTRACT

Objective: This case report describes a five years old boy that was brought to the emergency room by police in an unconscious state with suspected acute carbon monoxide poisoning. After initial management with 100 % oxygen through mechanical ventilation, patient showed remarkable improvement in neurological status. Three days after initial recovery, he showed abrupt and profound neurological deterioration indicating onset of delayed encephalopathy. The patient was managed again by 100% oxygen through mechanical ventilation and other supportive measures. He was exubated on day 10th and discharge after two weeks. With intensive physiotherapy and rehabilitation, there was nearly complete recovery after six months follow up.

***Keywords*:** Carbon monoxide poisoning, delayed encephalopathy

INTRODUCTION

Carbon monoxide (CO) is a colorless, odorless, toxic gas produced as a by-product of incomplete combustion of carbon-based fuels. It is a major indoor pollutant in the developing world and an important cause of mortality worldwide [1, 2]. Major sources of CO are household fires, motor vehicles, heater appliances [3].

Due to varying presentation of CO poisoning, ranging from vague flu-like symptoms to profound central nervous system dysfunction and prominent neuropsychiatric manifestation, it has been called "the disease of thousand faces" [4].

CO binds reversibly to hemoglobin with an affinity 200-250 times that of oxygen, thereby blocking the capacity of hemoglobin to transport oxygen leading to oxygen debt and lactic acid accumulation. The brain and myocardium are more susceptible to hypoxia due to increased oxygen demand [5].

Majority of the patients of CO poisoning recover well without any complication with hyperbaric or high oxygen therapy. However the incidence of delayed neuropsychiatric sequelae is seen in 10-30% of these cases [6].

The clinical presentation of delayed encephalopathy is in the form of deterioration and relapse of cognitive ability, behavior movement after initial recovery from acute CO poisoning [7, 8]. Latent period between acute presentation and delayed encephalopathy may vary from few days to weeks [8].

The most common grey matter lesion is bilateral necrosis of the globus pallidus. Other parts like hippocampus or focal area of cortex may also be affected [8].

A case of delayed encephalopathy in five years old boy following CO poisoning is reported here. It was characterized by apparent recovery after acute poisoning, followed by an abrupt and profound neurological deterioration with a seemingly reversible course. Diffusion weighted magnetic resonance imaging was quite characterstic in our patient.

CASE REPORT

A five years old boy was brought to the Pediatrics Emergency unit of Alnoor Specialist Hospital on 20/8/1432 by police after evacuating him from a burning building. Parents after evacuation were admitted in the medical unit of other nearby hospital. It was not known how long the patient has been exposed to the toxic fumes before police team arrived. The source of the fire was short circuiting of electrical wires in the kitchen which happened late in the night when family members were sleeping. He was brought in

unconscious state (GC scale 8/15) with irregular breathing, poor peripheral pulses, delayed tissue perfusion and hypotension (BP 80/40 mm of Hg). His mucous membranes were pink. There was smell of smoke but no thermal injury or burn. Evaluation of central nervous system revealed an unconscious child with normal sized pupils reacting to light, head lag, generalized hypotonia, generalized hypereflexia, power of 2/5 in all the four limbs and bilateral positive babinski.

His initial arterial blood gas (ABG) showed pH: 7.10, PCO_2: 38 mm, PO_2:40 mm of Hg, O_2 saturation 60%, HCO_3: 12.8, SBE: – 16. He was immediately intubated and connected to mechanical ventilator with 100% oxygen. He was sedated with fantanyl and medozolam. He received bolus of normal saline and started on dopamine. He was put on broad spectrum antibiotics, prophylactic phenytoin and ranitidine. He was also started on dexamethasone for presumed brain edema. Repeat ABG within half an hour showed improvement, pH: 7.32, PCO_2: 35.6, PO_2: 68 mm, HCO_3: 17.8, SBE: -8. His tissues perfusion and BP also improved (110/70mm of Hg).

His other initial investigations revealed CPK: 3176 IU/L, CK MB 77 IU/L and LDH of 396 IU/L. His CBC, ESR, serum electrolytes, liver enzymes, blood sugar, urea and creatinine were essentially normal. Electrocardiogram upon admission showed normal sinus rhythm with no other abnormality. Skeletal survey did not show any fracture. Initial brain CT scan was normal. Due to absence of appropriate facilities, carboxyhaemoglobin (COHb) measurement could not be carried out.

There was remarkable progress in next 4 days. He regained consciousness. He was exubated and did well after extubation. His ABGs & vitals were maintained within normal range. He started moving upper limbs and lower limbs with good tone and reflexes.

On 6th day of admission, his condition deteriorated. He developed generalized tonic and clonic convulsions and became unconscious. Examination at this point revealed GC scale 8/15, BP 119/71, HR 110/m, RR 25/m, generalized hypotonia, hypereflexia, grade 2/5 power in all four limbs and bilateral positive babinski. There were no meningeal signs and pupils were bilaterally dilated but reacting to light. He was again shifted to ICU and ventilated with 100% O_2, PIP/PEEP 28/6. Because of his progressive neurological problems, further investigations were carried out. CSF analysis was done which showed RBC: 1000/mm WBC: 5/mm, protein 26mg%, sugar 116 mg%, & sterile culture.

MRI brain revealed swollen basal ganglion displaying high signal intensity on FLAIR and T2 WI. On diffusion WI, centrum semiovale displayed restricted diffusion. Considering the patient's history, this finding was suggestive of global ischemia due to CO poisoning.

He was continued on mechanical ventilation, started on IV phenbarbitone, dopamine. He showed remarkable improvement and exubated on 10th day. In-patient physiotherapy and intensive rehabilitation was started and he was discharged after 2 weeks. At the time of discharge he was fully oriented, responsive, moving all the four limbs but still could not sit in the bed. He was advised to continue physiotherapy at home.

On the last follow up in out-patient clinic after six months, he was able to ambulate without any assistive devices.

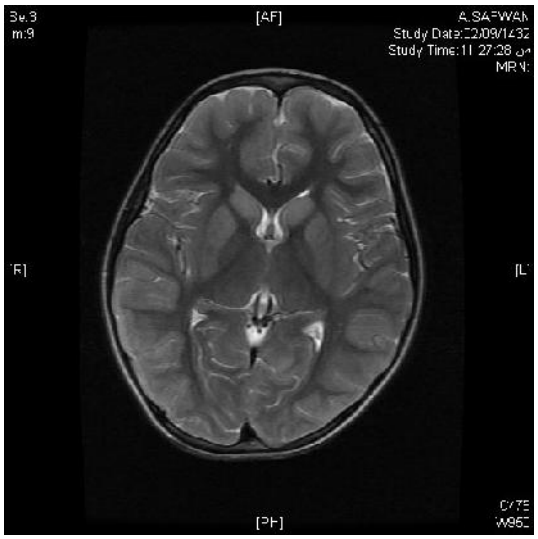

Figure 1

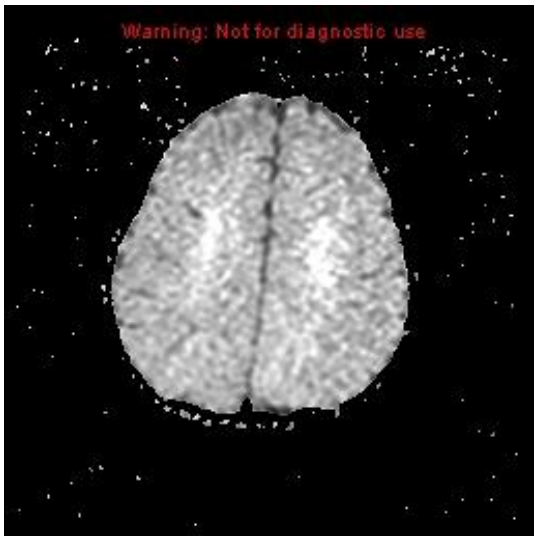

Figure 2

DISCUSSION

In the patient under report, positive history of exposure to fumes, convergence of symptoms and signs along with characterstic brain MRI findings satisfied the criteria for the diagnosis of CO toxicity, even in absence of blood COHb level. The studies conducted have shown significant correlation between severities of toxicities with neurological manifestation than with blood CO level, thus validating the importance of clinical judgment [9, 10]. However, it is important to determine blood CO level to confirm the diagnosis if facilities are available.

The classical presentation of delayed encephalopathy includes apathy, disorientation, amnesia, extra-pyramidal symptoms, incontinence, psychosis, global cognitive impairment, seizure, and coma [11].

Patients with delayed encephalopathy due to CO poisoning usually have a lucid period of few weeks before the appearance of a progressive decline in their neurocognitive functions [7, 8]. However in our patient the lucid interval was very short (3 days) and clinical presentation was profound in the form of seizure, coma and quadriplegia. The severe presentation in our patient may be because of his young age.

Functional imaging has been used to show globus pallidus, basal ganglion to be one of the most frequently injured areas during the acute stage of CO poisoning [8]. MRI in our patient revealed swollen basal ganglion displaying high signal intensity on FLAIR and T2 WI.

Recovery from delayed encephalopathy occurs in 50% -75% within one year [3, 12]. Risk factors predicting the onset of delayed encephalopathy in patients with acute CO poisoning is not so clear. Clinical status or carboxyhaemoglobin level on initial presentation could not predict the occurrence of delayed encephalopathy. Some investigators have shown a significant correlation between cerebral white matter changes on initial CT scan and the development of delayed encephalopathy in acute CO poisoning [13]. Brain MRI showing bilateral symmetric white matter hyperintensity (T2WI/ FLAIR) could be a good predictor of delayed encephalopathy in patient with acute CO intoxication [14]. Other imaging

technique like positron emission tomography and single photon emission tomography which detect the blood flow abnormality in affected area, might be more sensitive and better predictor of clinical course in patients with acute CO poisoning [15, 16].

The pathogenesis of the delayed encephalopathy of CO intoxication remains debatable; No single reason (e.g. hypoxia) alone is adequate enough to explain the varying presentations. Various theories have been proposed for this e.g. immuopathological damage, disturbance of dopaminergic and serotonergic functions, role of CO as an endogenous neurotransmitter[15].

Oxygen therapy including the use of hyperbaric oxygen has been for years the mainstay treatment of acute cases. Administration of 100% O_2 competitively displaces CO from its transport site of hemoglobin molecule, thereby decreasing the half life from 200-300 minutes to 60-90 minutes. Half life can be further shortened to 30 minutes by use of hyperbaric oxygen leading to quick removal of CO from the blood and thus preventing lipid peroxidation of the brain[5, 17]. Despite this convincing hypothesis, there are conflicting reports on the use of hyperbaric oxygen in acute CO poisoning.

A significant higher neurological morbidity was found in those treated with normobaric oxygen compared to those who received hyperbaric oxygen[18]. On the contrary, in a randomized controlled trial, Scheinkestel et al [19] compared the two different modalities and found poorer outcome in those treated with hyperbaric oxygen as compared to the group treated with normobaric oxygen.

CONCLUSIONS

This case report demonstrates that while treating the immediate complications of CO poisoning, clinician must be aware of delayed complications among survivors which usually occur few days to weeks after acute poisoning. Regular follow up and monitoring of neurocognitive functions of these patients is very critical to caution them and their families of this potential complication.

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