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CARBON MONOXIDE POISONING WITH INTRA UTERINE DEATH

Medicine

Dr Jerry Jacob Junior Resident, Department of Emergency Medicine St. John's Medical College Hospital, Bengaluru

Dr Karthik Reddy C H* Assistant Professor, Department of Emergency Medicine St. John's Medical College Hospital, Bengaluru *Corresponding Author

Dr Shakunthala Murty HOD, Department of Emergency Medicine St. John's Medical College Hospital, Bengaluru

ABSTRACT

Carbon monoxide is a colourless, odourless toxic gas produced as a by-product of incomplete combustion.The symptoms of carbon monoxide poisoning are nonspecific;therefore high index of suspicion is required for diagnosis. Here we present a case of 25 year old female presented to ED with an obstetric score of G2P1L1 at 6 months of gestation with a history of being found in semiconscious stateat home by neighbours. On examination she was tachycardic with a BP of 140/70 mmHg and was hypoxic.Patient was drowsy,bilateral diffuse crackles present on auscultation and absent foetal heart sounds. Bedside USG revealed bilateral B lines and ECHO showedleft ventricular systolic dysfunction with EF of 30%. Arterial blood gases with co-oximetry revealed carboxyhaemoglobinemia with COHb of 14%. The bedside USG showed no fetal cardiac activity. A final diagnosis of Carbon monoxide poisoning with IUD was made. The husbandand son also presented with altered mental status and respiratory distress with elevated COHb levels. On repeated history taking it was revealed that they procured LPG (liquid petroleum gas) from a local vendor and that incomplete combustion of the cheap LPG lead to the carbon monoxide poisoning.

KEYWORDS

Carbon monoxide poisoning, IUD, Co-oximetry, carboxyhaemoglobin.

INTRODUCTION

Carbon monoxide (CO) is a colourless, odourless, tasteless and non-irritant toxic gas. It is usually produced by incomplete combustion of hydrocarbons, wood, charcoal and coal.Carbon monoxide has 210 times greater affinity for haemoglobin and 60 times for myoglobin than oxygen. Carbon monoxide exposure has an especially deleterious effect on pregnant women. Very fewcases of carbon monoxide poisoning with IUD have been reported so far.Normal levels of CO in adults is <2% and in smokers, upto 6-8%. Any values above this levels is considered toxic.

CASE REPORT

25 year old female patient with 6 months of amenorrhea,LMP being 20thJan 2018,presented to ED with history of breathing difficulty and altered sensorium.In the morning patients neighbour tried waking her up by knocking the door, as there was no response to phone calls. As the door wasn't opened on knocking, they broke it downto find her, her husband and son in semi-conscious state on the floor and was rushed to the hospital. Patient had history of consumption of corn the previous night at 8.30 pm prior sleep.No alleged h/o deliberate ingestion of any substance with the intention of DSH. No significant past history.

On examination-patient is drowsy with a BP of 140/80mmHg,PR-140bpm,RR-30cpm,Spo2-84% on RA(100% with NRBM).Bilateral diffuse crackles present on auscultation, bilateral plantar is flexor and moving all four limbs, pupils-b/l 3mm reactive to light. Bedside Lung USG revealed bilateral B lines and ECHO showed EF of 30%(LV Systolic dysfunction).Abdomen USG revealed no fetal cardiac activity.ECG(Figure 1) showed sinus tachycardia.Arterial blood gas showed pH 7.28, $PaCO_2$ 45.5, PaO_2 80.4, HCO_3 20.8,COHB-14%, SaO_2 95%(with 10 ltrs of oxygen through NRBM).In view of worsening respiratory distress patient was intubated and was placed on high PEEP.CXR(Figure 2) suggestive of bilateral lung infiltrates(acute pulmonary edema secondary to LV dysfunction).Patient was treated with diuretics,antibiotics, Mechanical ventilation with FiO2 100%,PEEP 10cm H_2O.Dead fetus expelled out after two days of presentation. Patient was extubated after 5 days of treatment in ICU and ECHO done before discharge showed normal biventricular function with EF-67%, no RWMA.

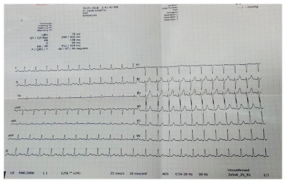

Figure 1:

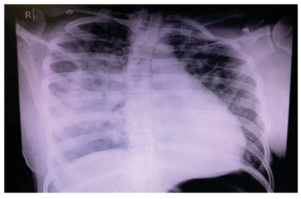

Figure 2:

DISCUSSION

Carbon monoxide is a fatal poison.It is produced by incomplete combustion of any hydrocarbon (gas,coal,charcoal, petrol, diesel,paraffin) or wood.CO binds to haemoglobin in the blood with high affinity, forming COHb(1-4). CO binds with high affinity to many ferrous heme-containing proteins. Hb has a 250-fold greater affinity for CO than for oxygen (5-7). CO competes with oxygen for binding to Hb and by displacement of oxygen, reduces oxygen carrying capacity.

CO inhibits mitochondrial respiration by binding to COX, effectively shutting down oxidative phosphorylation (8-11). Excess CO activates platelets by displacement of NO from platelet surface (8). Displaced free NO can react with superoxide to produce peroxynitrite, further inhibiting mitochondrial function and increasing platelet activation (8-11). Activated platelets can stimulate neutrophils (11,12) to degranulate and release myeloperoxidase (MPO) (11). MPO amplifies the inflammatory effects by triggering more neutrophil activation, adhesion, and degranulation (12)

The clinical presentation of carbon monoxide poisoning ranges from headache, giddiness,chest pain,ataxia,seizure,bullous skin lesions, focal neurologic deficit, coma and death.The cardiovascular effects of CO include myocardial ischemia, arrhythmia and stunned myocardium syndrome.CO poisoning is diagnosed by a clinical triad of (*1*) symptoms consistent with CO poisoning; (*2*) history of recent CO exposure; and (*3*) elevated COHb levels (13). Diagnosis should be confirmed by measurement of elevated COHb levels in blood (14). Conventional pulse oximetry cannot distinguish between COHb and oxyHb, and, as such, can miss significant COHb levels and profound hypoxia (14). Pulse CO oximetry can measure multiple species of Hb (COHb and methemoglobin) by using readings at eight wavelengths of light instead of the two wavelengths used by standard oximetry, which can measure only deoxyHb and oxyHb. Elevated lactate, troponin, creatine kinase and elevated anion gap metabolic acidosis may be seen in carbon monoxide poisoning.

Current therapy for CO poisoning is 100% normobaric oxygen (NBO_2) or HBO_2 (2.5–3 atmospheres) (15). NBO_2 and HBO_2 remove CO at a faster rate from the blood by increasing the partial pressure of oxygen, which increases the dissociation rate of CO from Hb (15-16).HBO_2 should be considered for all cases of serious acute CO poisoning, including syncope,altered mental status ischemic cardiac changes, pregnancy with COHB levels >15%,neurological deficits, significant metabolic acidosis, or blood COHb > 25% (17).

FOOTNOTES

There is no conflict of interest

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