Cardiac Manifestations Of Carbon Monoxide Poisoning

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Introduction

Carbon monoxide (CO) poisoning is one of the most critical health concerns worldwide due to severe clinical effects with high morbidity and mortality. Tissue hypoxia and cellular damage from CO poisoning results in oxidative stress. Organs and tissues with high O2 demand including central nervous and cardiovascular system are more susceptible to this oxidative stress. We investigate and describe the cardiovascular manifestations in 350 patients who were admitted for moderate to severe CO poisoning and treated with hyperbaric oxygen (HBO2) therapy.

Objective

Identify the common cardiovascular manifestations of carbon monoxide poisoning.

Methods

We retrospectively collected data on 350 consecutives adult patients treated for CO poisoning between January 2011 to April 2018 at Advocate Lutheran General Hospital (ALGH). Cardiac biomarkers, EKG, carboxyhemoglobin (COHb) levels were obtained from pre-hospital sources including EMS (emergency medical services), outside hospital at the time of transfer or at ALGH. Glasgow Coma Scale (GCS) measurements were obtained at initial pre-hospital evaluation and upon arrival to ALGH. Patient demographics, cardiovascular, cerebral vascular diseases history and cardiac risk factors including hypertension (HTN), hyperlipidemia (HLD), diabetes (DM), smoking history were obtained from electronic medical record. Myocardial injury was defined by cardiac troponin I level of >0.05 ng/mL.

Results

Characteristics of patients admitted for carbon monoxide poisoning

Figure 1. shows patients characteristics of 350 patients admitted for CO poisoning. The mean age was 47.3 years with 60% men; 89% of the admission were accidental exposure. In terms of cardiac risk factors, 18% were active smoker, 16% HLD, 25% HTN, 11% DM, 2% had all four cardiac risk factors. Additionally, 2% had previous revascularization. On admission 15% were intubated for airway protection.

Figure 2. Flow chart shows data extractions from EMR of 350 patients admitted for carbon monoxide poisoning. 303 patients had EKG completed on admission, out of that 223 patients had troponin data available. 88 of 223 patients had elevated troponin, with 67 out of 88 had TTE. Out of which 16 had wall motion abnormality and 8 underwent coronary angiography evaluation.

Figure 3. Pie chart shows EKG characteristics of 87%(303/350) of the sample population. Majority had a normal sinus rhythm (NSR). Diagnostic ischemic changes on EKG defined has ST or T wave changes noted in 5.9%(18/303). Additionally, 20.8% (63/303) sinus tachycardic, 1.7%(5/303) prolonged Qtc. No EKG data available on 13%(47/350).

Outcomes

Pie chart shows final outcomes. Mortality 1.7% with majority PEA arrest. Additionally, 8 patients with abnormal TTE underwent coronary angiography evaluation, 2/8 went for PCI, 2/8 bypass surgery evaluation with one patient who refused surgery. 4/8 with no significant coronary disease.

Discussion

Myocardial injury is common and widely seen with CO poisoning as seen by elevated cardiac biomarkers in 40% of the tested population. Patient with CO poisoning should undergo evaluation with an EKG and serum biomarkers. If an abnormality is detected, patient should undergo an echocardiogram. Further evaluation with angiography may be warranted in patients with left ventricular dysfunction and underlying coronary artery disease especially in patients with risk factors.

References


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