

Breathanalysis of expired carbonmonoxide and its correlation with symptoms of toxicity among urban automobile drivers

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Abstract

Introduction: Automobile emissions form the leading cause of air pollution in urban areas. Carbonmonoxide (CO) is one among the six major pollutants of air caused by vehicles. Because it is colourless, odourless and tasteless gas, exposure can cause symptoms which can be unobserved. Drivers are at risk as exposure is maximum, posing an occupational hazard.

Aim: To screen the urban automobile drivers for exhaled breath Carbonmonoxide (eCO) and to correlate eCo with symptoms experienced that may be due to CO exposure.

Materials and Methods: A cross sectional observation of Breathanalysis of automobile drivers (n=101) for expired CO (eCO) was done using CO Breathanalyser (Vitalograph 29700). Data regarding age, duration of exposure, hours spent in driving per day, other sources of exposure such as smoking status, history of passive smoking, exposure to coal fire in the household, place of work were obtained. Symptoms pertaining to CO exposure like headache, body pain, tiredness, and visual disturbances like irritation of eye, redness, neurological and preexisting medical condition were analyzed for correlation with eCO.

Results: Mean expired carbonmonoxide, eCO in breath was 2.45 ppm in the study group of urban drivers with mean age 39.96. In 87% of the drivers, eCO was 6ppm. 12.9% of drivers had more than 6 ppm of eCO. 22.8% of drivers exhibited eCO between 3-4 PPM. Analysis of symptoms revealed, tiredness (27%), eye symptoms like irritation and redness (10%), headache (20%), body pain (8%) & fatigue (7%). Significant correlation was observed with body pain (P = 0.04) and fatigue (P=0.03).

Conclusion: Urban drivers are regularly exposed to immense automobile vehicle exhaust which can inflict carbonmonoxide toxicity and impair working capacity.

Keywords: Urban air pollution, Drivers, Carbonmonoxide, Breathanalysis.

Introduction

Carbonmonoxide is classified as primary pollutant as well as critical pollutant of air. The chief source of carbonmonoxide in air is purely anthropogenic contribution, resulting from incomplete combustion of fossilfuel, tobacco smoke, fumes from factories and industries, vehicle emissions, leaking furnaces and chimneys, automobile exhaust from attached garages etc.¹ Because the physical nature of the gas being colourless, odourless, and tasteless, it is often termed as "silent killer". Exposed individuals suffer toxic symptoms quietly and quickly depending upon the type of exposure. Carbonmonoxide can induce immediate health effects and even be fatal at high concentrations. At low concentrations, CO exposure leads to moderate cardiovascular and neurobehavioral adverse effects and in significantly high concentrations symptoms like unconsciousness and death occur after acute or chronic exposure.^{2,3}

CO has a high affinity about 250 times that of oxygen inducing shift of oxyhemoglobin dissociation curve to the left resulting in anemic hypoxia. Tissue hypoxia further worsens when CO binds to myoglobin resulting in cardiac depression. At the cellular level oxygen uptake is blocked due to binding of CO to cytochrome oxidase. Hypoxia induced reperfusion generates a free radical, peroxyxynitrate capable of lipid peroxidation. Mitochondrial dysfunction, apoptosis and leucocyte sequestration are the pathological

changes reported in brain structures such as striatum, hippocampus, cerebral white matter and cerebellum.⁴ Cardiovascular and neurological manifestations are usually encountered in patients diagnosed with CO poisoning.

Exposure to low concentrations of CO as in occupations like drivers, traffic personnel and factory workers that may occur periodically, produce long term health effects inducing morbidity and mortality.⁵ Symptoms experienced initially due to exposure can generally present with flu like symptoms or it often leads to misdiagnosis of food poisoning.⁶ Mild non-specific symptoms such as lethargy, headaches, fatigue, visual disturbances, and body pain are usually the presenting symptoms due to CO toxicity which depends on the concentration of CO.⁷ Neurological manifestations like confusion, dizziness, amnesic syndromes, dementia, psychosis, Parkinsonism and delayed neurological sequelae are observed due to chronic exposure.^{8,9} Levels of CO in ambient air have been correlated with mortality rates and increase in rate of hospitalization for heart failure as per previous reports.¹⁰ Co-morbid conditions can worsen due to effects of CO exposure evidently.

Rapid urbanization with increasing vehicle density annually, in the recent past promotes air pollution to a great extent. As per WHO reports on latest urban air quality database, annual emission of Carbonmonoxide is immensely high in Indian urban areas.¹¹ Drivers are prone to prolonged

exposure from vehicle exhaust regularly that might increase the chance of CO toxicity. The working capacity can be impaired among drivers owing to exposure at various levels in the work place producing diverse effects such as headache, lethargy, coma and death.¹² Reported cases of occult CO poisoning due to faulty automobile exhaust system among drivers are also available.¹³ Automobile drivers are at greater risk of suffering from symptoms like headache, palpitation, vertigo, irritability than the non-exposed drivers and also greater audio-visual reaction time making them more prone for traffic accidents¹⁴ As the symptoms can present diversely, misdiagnosis of CO poisoning is quite frequently made. As poor correlation exists between the levels of Carboxyhemoglobin (COHb) and the signs, symptoms at presentation, the diagnosis and prognosis of CO poisoning becomes more complex.¹⁵ Devices that measure expired CO in breath can be of immense use in suspected cases of CO poisoning. Breathanalysers that were initially used for smoking cessation to measure eCO can aid in the diagnosis of CO toxicity.¹⁶

Automobile drivers in urban areas are prone for occult CO poisoning owing to periodic exposure in varying concentration. Workplace accidents ensue among exposed drivers compromising the safety. Neurological symptoms such as reduced concentration, memory disturbances and reduced cognitive performances can arise because of toxic exposure.¹⁷ Previous reports on ambient levels of CO, COHb of the exposed are available in other areas of the globe.¹⁸ Limited observations prevail among automobile drivers of Indian urban areas involving breathanalysis of carbonmonoxide.¹⁹ This study was hence, designed to measure the exposure status of urban automobile drivers to toxic effects of Carbonmonoxide using breathanalyser.

Materials and Methods

A cross sectional analysis of expired breath Carbonmonoxide was conducted among urban drivers after approval by Institute Ethics Committee. Automobile drivers (n= 101) using different type of motor vehicles in urban Chennai were enrolled after obtaining informed consent.

Heavy traffic zones in Chennai were identified for sample collection of breath analysis of CO from automobile drivers after obtaining informed consent. Four different locations such as bus depots of Chennai, where there is exceedingly high vehicle population were considered for study area. Sampling was done in the evening between 4 pm to 6 pm to maintain uniformity. A single breath sample was collected into a hand held breath CO analyser (Vitalograph 29700).

Data regarding age, duration of exposure, hours spent in driving per day, other sources of exposure such as smoking status, history of passive smoking, exposure to coal fire in the household, place of work were obtained. The subjects in our study were categorized into various groups based on their age, duration of experience, smoking status, type of vehicle used, type of fuel used and any symptoms pertaining to CO toxicity. Symptoms pertaining to CO

exposure like headache, body pain, tiredness, and visual disturbances like irritation of eye, redness, neurological and preexisting medical condition were analyzed for correlation with expired breath Carbonmonoxide

1. Inclusion criteria included apparently healthy urban drivers of age group 18-60 years.
2. Urban drivers of age more than 60 years, pre-existing respiratory disorder, acute URI or LRI, other systemic illness like Diabetes, Hypertension, and Ischemic heart disease were excluded.

Breath CO analyser (Vitalograph 29700) which is portable, rapid and non-invasive device was used. It measures breath CO levels in parts per million (ppm) based on the conversion of CO to carbon dioxide (CO₂) over a catalytically active electrode. Widespread use of such non-invasive analysers can supplement diagnosis in suspected cases.²⁰

Data collected were analyzed using SPSS version 23. Pearson's correlation coefficient was used to examine the relationship between eCO and symptoms recorded. Group analyses were performed using ANOVA for quantitative variables. P value less than 0.05 was considered to be statistically significant.

Results

Drivers of different automobiles such as auto rickshaw (30.7%), bus (55.4%), car (4%), maxi-cab (6.9%), and van (3%) were participants of our study. Fuels used by the automobile drivers include diesel 72% (Mean eCO=3.01 ± 0.38), petrol 8% (Mean eCO=5.12±2.72) and gas 21% (Mean eCO=6.8 ±1.7). The mean age of the study group in years is 39.96, of which 17.8% were less than 30 and 13.9% were above 40 years of age. Mean eCO in expired air among the drivers was 2.5 ppm. 87% of the drivers had eCO<6 ppm. 12.9% of drivers exhibited eCO>6 ppm. 22.8% of drivers exhibited breath eCO between 3-4 PPM (Table 1). Mean length of exposure taken as duty hours per day among the drivers was 12 and mean experience in years was 14.68.

Table 1: Percentage distribution of eCO (ppm) among urban automobile drivers

eCO (ppm)	Percentage %
> 6	12.9
< 6	87
3-4	22.8

Analysis of symptoms revealed tiredness (27%), visual symptoms like irritation and redness (10%), headache (20%), body pain (8%), fatigue (7%), palpitations (1%), chest pain (2%) (Fig. 1). Mean eCO in drivers with tiredness (4.64 PPM), redness of eye (4.3 ppm), headache (4.35 PPM), body pain (4.16 PPM) and fatigue (7.66 PPM). Correlation was observed with body pain (P = 0.04) and fatigue (P=0.03), which were statistically significant. eCO was also significantly associated with duration of exposure (Mean=14.68, P= 0.26). No correlation was observed with age and experience.

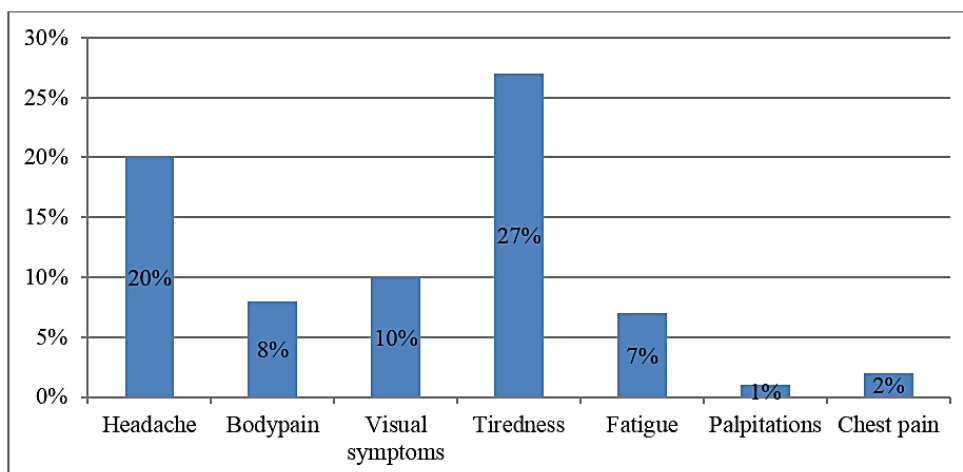


Fig. 1: Symptom analysis due to CO exposure among urban automobile drivers

Discussion

Our cross-sectional observation of breath analysis of expired Carbonmonoxide (eCO) among the exposed group of urban automobile drivers was within the observed standard limits.^{21,22} Mean breath Carbonmonoxide as per our study and COHb equivalent are 2.45 ppm and 0.68% respectively. However, 12.9% of the drivers exhibited eCO above 6 PPM, which is considered as the cutoff limit for toxicity.²³ Reported normal levels of COHb are about 0.83% and classic symptoms such as headache and dizziness are observed with COHb levels ranging between 10%-30%. However, toxic symptoms do not always correspond to COHb.^{24, 17}

Traffic rich zones can act as point source and constant mobility of vehicle users can be considered as line source in terms of air pollution. Both of which can add to the toxicity produced. Various factors like the concentration of toxic CO in ambient air, physical determinants like humidity, temperature, level of physical exertion are some of the factors which can alter the outcome.²⁵ The type of vehicle, fuel used and duration of experience can also modify the expired level of eCO significantly according to our previous report.²⁶ Ambient air exposure to CO of 10 PPM for 8 hours can result in hazardous limit of COHb 2.5% as suggested by Expert Panel on Air Quality Standards.²⁷

Symptoms that are significantly correlated in our study include body pain and fatigue. Symptoms of CO exposure typically, were observed on regular basis with recovery after removal from the source. Fatigue can be possibly explained by the resulting hypoxia due to exposure to CO, which has a profound effect on decreasing the resistance to fatigue in skeletal muscle.²⁸ Regular occurrence of toxic symptoms followed by recovery after removal of the source is the rule reported in previous studies.²⁹ Low dose of CO exposure for a prolonged period can produce subtle neurological symptoms whereas transient exposure may not have similar effect.^{30,31} Our study group involved apparently healthy drivers and symptoms reported were non-specific, regular in occurrence with recovery upon removal of the source. Thus, exhibited symptoms may be attributed to low dose

exposure. The half-life of inhaled CO ranges from 2 to 6 h which can vary due to other determinants like respiratory rate.^{25,32} Headache was present as a symptom in 20% of the respondents however, not significantly correlated with expired CO. Previous reported findings have correlated headache and COHb levels which was considered as alerting symptom of CO poisoning.³³

Prevailing reports have suggested the disparity between the estimated COHb and the severity of toxicity. Hence, it may go unnoticed if COHb alone is taken into account in suspected cases. Chronic exposure and sub-acute poisoning with CO can produce neuropsychological disturbances in memory, sleep, vision, olfaction, psychomotor activity as reported in few identical cases which may interfere with the driving skills compromising safety of the drivers.^{34,35} Prevalence of higher vehicle density in Chennai with Air quality index of 59 and prominent pollutant as Carbonmonoxide paves way for urban automobile drivers to these toxic effects of Carbonmonoxide.

In view of the obscuring symptomatology due to CO exposure, toxicity remains unobserved and may even be fatal. Breathanalysis for eCO, being a simple technique can be routinely employed in suspected persons who are occupationally exposed and present with vague symptoms to aid in early and prompt diagnosis.

Conclusion

Urban automobile drivers are at constant risk to exceed the toxic limits of carbonmonoxide exposure. Factors like duration of exposure, concentration of CO determined by type of vehicle and fuel used, physical factors and existing morbidity can influence the toxicity. Symptoms due to toxicity are so diverse and can often be underreported and unrecognized among professionally exposed drivers. Health education on CO toxicity, careful monitoring of ambient CO in vehicles may be advocated to prevent toxicity.

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Conflict of Interest: None.

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