

Toxicological Investigation of Acute Carbon Monoxide Poisoning in Four Occupants of a Fuming Sport Utility Vehicle

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ABSTRACT

Background: This toxicological investigation involves a report on the death of four occupants of a sport utility vehicle on one of the major busy Federal roads of Nigeria where they were held for up to three hours in a traffic jam while the car was steaming.

Methods: Autopsy was executed using the standard procedure and toxicological analysis was done using simple spectrophotometric method to establish the level of carboxyhaemoglobin (HbCO) in peripheral blood in the four occupants.

Results: The autopsy report indicated generalized cyanosis, sub-conjunctival hemorrhages, marked laryngo-trachea edema with severe hyperemia with frothy fluid discharges characteristic of carbon monoxide poisoning. Toxicological report of the level of HbCO in part per million (ppm) in the peripheral blood of the four occupants was A= 650 ppm; B= 500 ppm; C= 480 ppm, and D= 495 ppm against the maximum permissible level of 50 ppm.

Conclusion: The sudden death of the four occupants was due to excessive inhalation of the carbon monoxide gas from the exhaust fumes leaking into the cabin of the car. The poor road network, numerous potholes, and traffic jam in most of roads in Nigeria could have exacerbated a leaky exhaust of the smoky second hand SUV car leading to the acute carbon monoxide poisoning.

Keywords: Acute Poisoning, Autopsy, Carbon Monoxide, Toxicological Studies.

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INTRODUCTION

Carbon monoxide (CO) is a colorless, odorless gas produced by incomplete combustion of carbonaceous material (gasoline, propane, natural gas oil, wood, coal, tobacco, or charcoal). CO is produced often in domestic or industrial settings by older motor vehicles and other gasoline powered tools, heaters, and cooking equipment. CO is formed as a by-product of burning organic compounds. Most fatalities, however, result from fires; stoves, portable heaters, and automobiles exhaust which cause approximately one third of deaths. CO poisoning is often associated with malfunctioning or obstructed exhaust systems and suicide attempts. Cigarette smoke is also a significant source of CO. Natural gas contains no CO, but improperly vented gas

water heaters, kerosene space heaters, charcoal grills, hibachis, and Sterno stoves all emit CO [1].

Other causes of CO exposure include propane-fueled forklifts, gas-powered concrete saws, and indoor tractor pulls, as well as inhaling spray paint and swimming behind a motorboat. Industrial workers at pulp mills, steel foundries, and plants producing formaldehyde or coke are at risk for exposure, as are personnel at fire scenes and individuals working indoors with combustion engines or combustible gases. CO intoxication also occurs by inhalation of methylene chloride vapors, a volatile liquid found in degreasers, solvents, and paint removers. A significant percentage of methylene chloride is stored in the tissues and its continued release results in elevated CO

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levels for at least twice as much as with direct CO inhalation. Children riding in the back of enclosed pickup trucks seem to be particularly at a high risk [1, 2].

Carbon monoxide poisoning occurs after inhalation of the gas. Since it is a tasteless, non-irritating gas, it is difficult for people to detect. It is in most cases a product of incomplete combustion of organic matter due to insufficient oxygen supply to convert it to carbon dioxide. Exposure at 50-100 parts per million (ppm) or greater can be dangerous to human health [1]. However, many cases of sudden deaths resulting from pollution of smoke of electricity generators used as alternative source of power in Nigeria have been reported [2].

This study is a unique investigation of a particular case of interest involving the death of four occupant of a Sport Utility Vehicle (SUV) who died following inhalation of carbon monoxide gas produced by leaking exhaust fumes of the car with partially opened boot. This report is exceptional in that all occupants (males) in their middle ages were talking to each other but suddenly slept off quietly while the car was still steaming in the traffic jam.

CASES

This is a case of four friends travelling in a second hand SUV for an occasion. On their way, they faced a heavy traffic jam following the chain accident of long haulage vehicles that sparingly blocked a larger part of the road. These resulted in the blockage of on-coming and out-going traffic. This hold up made them open their "SUV" boot partially to let in oxygen in the very humid climate of the season. Initially, they were talking and arguing but as the jam persisted tiredness ensued with consequent death of all the occupants. As the traffic started moving, the immediate vehicle behind, in an attempt to move, honked his horn but there was no response. He walked out to beckon on them to move only to see no response from the drowsy and sleepy men. The vehicle was moved off the road and the dead occupants were taken to a nearby hospital. They were certified dead by the medical team on duty and full investigation was ordered.

MATERIALS AND METHODS

Materials

Heparinized cuvette, blood samples, oxyhaemoglobin (HbO₂), and carboxy hemoglobin (HbCO) standards were prepared using the standard procedure.

This study started with coronary inquest in which a postmortem examination was executed to ascertain the cause of sudden death of all four middle-aged male occupants in an SUV on one of the major highways of Nigeria where they were held for up to three hours by traffic jam while the car was steaming with a partially opened boot.

Gross Anatomical and Pathological Examination

Histopathological evaluation was carried out on the four bodies and their major organs using standard autopsy procedure.

Toxicological Analysis

Toxicological analysis of HbCO from peripheral blood was done using simple spectrophotometric method as described by Katsumata *et al.* [3]. The determination was carried out using Hitachi 557 spectrophotometer (Hitachi, Ltd., Tokyo, Japan). Solid sodium hydrogen sulphite (2mg) was added to 2.5mL of 1% sodium carbonate in a cuvette. This was followed by addition of 10µL of blood and 0.2 mL of 5N sodium hydroxide with mixing after each addition. After standing for 5 minutes the absorbencies were read at 558 nm (A₅₅₈) against water as blank. The final results of HbCO for the four deceased were estimated in part per million.

RESULTS

The autopsy reports and the spectrophotometric assay of HbCO in peripheral blood are presented as follows:

Autopsy Findings (Four Middle Aged Males)

There was generalized cyanosis of the bodies with sub-conjunctival haemorrhages. The facial area showed multiple discrete petechial haemorrhages. There was marked laryngo-trachea edema with severe hyperemia with frothy fluid discharges. The lungs were wet, shining with multiple discrete anthracitic patches and moderate to severe edematous. The organ weights are shown in Table 1.

Table 1. Organ weights (g) of samples from the four victims.

Organs	Organs weights (g) of samples from four victims				Organs normal maximal weight(g) in men
	A	B	C	D	
Heart	560	300	340	520	340
Liver	1000	900	1200	980	1400
Spleen	100	100	100	120	175
Kidney Right/Left	100/120	120/150	100/130	100/100	125/170

The hearts of 'A' and 'D' showed thickened anterior left ventricular wall measuring 1.8 cm and 2cm, respectively. The interventricular septum was thickened with atherosclerotic narrowing of three major coronary vessels up to 80% of the lumen. In the case of 'B' and 'C', all valvular markings were within the normal range. The brains of all showed a salmon pinkish colour with mild to severe cerebral edema. The cause of death for 'A' and 'B' was acute left ventricular failure due to hypertensive heart disease following carbon monoxide poisoning while 'C' and 'D' died due to acute laryngo-trachea edema following carbon monoxide poisoning.

Toxicology Analysis

Blood from the periphery was analyzed for carbon monoxide using the visible spectro-photometric method. The results are given below in parts per million (PPM).

Table 2. Determination of HBCO using samples from four victims (A-D).

S/No	Samples from four victims	Quantity of CO (ppm)
1	A	650
2	B	500
3	C	480
4	D	495

DISCUSSION

In the past, suicide by carbon monoxide poisoning occurred by mistakenly running a car engine in a closed space, such as a garage or by redirecting a running car's exhaust back inside the cabin with a hose. In these cases, the motor car exhaust may have contained up

to 25% carbon monoxide. However, catalytic converters found on all modern automobiles eliminate over 99% of carbon monoxide produced. Also, further complications from unburnt gasoline emissions can make exhaust fumes unbearable to breathe before losing consciousness [4].

However, the commonest cause of death in carbon monoxide poisoning is respiratory failure following laryngo-tracheal edema as seen in the US where generators have found usage during storms or in remote areas [5]. In all cases reported and even in this report, CO poisoning occurs following inhalation of this gas at a concentration above 50 ppm, as this is the maximum allowable carbon monoxide concentration for continuous exposure in healthy adults in an eight-hour period [6].

Death, therefore, ensues from exposure to high concentrations of CO due to the formation of carboxyhaemoglobin (COHb) which impairs oxygen carrying capacity of Hb. There has even been evidence of poisoning from CO to as low as 25 ppm in healthy individuals [7].

The sudden death of the four victims (A-D) are because they have already developed cardiac arrhythmias and electrocardiographic signs of ischemia which ensured lack of memory and attention and Parkinson-type altered movement. This is even worst in A and D patients with long standing history of coronary heart disease as 80% of coronary vessels had been compromised by arteriosclerosis and hypertension. This is the causes of the enlarged heart (Table1) which in part might have accounted for the sudden hypoxic state

of most of the tissues/organs and rapidity in death [8].

Kales [9] (1993) reported that the resultant effect of reduction in oxygen-carrying capacity can lead to end organ hypoxia with sudden death as the ultimate outcome.

The difference in perfusion of carbon monoxide in Table 2 may be due to the position where the individual was sitting in the car and possibly the difference in the rate of absorption/threshold levels.

It is important to note that lack of knowledge of the dangers of carbon monoxide poisoning was at play here; otherwise, hence they would not have opened the boot as to let in exhaust fumes of carbon monoxide while they were in the traffic jam.

This study indicates the need for close monitoring of cars in traffic jams by installing cheap battery operated carbon monoxide detectors as most car occupants are at a high risk of poisoning from exhaust fumes. Besides, in cars where there is solid fuel for heating, carbon monoxide detectors should be installed if not available [10].

CONCLUSION

This study is to make the public aware of the dangers of possible exposure to toxic doses of CO. Report of cases of intoxication with CO is on the increase in Nigeria. Three well diggers who lowered pumping machine on a support into a well to pump out water, met their untimely death when they attempted to clean up the well immediately after pumping out water. Besides, there is also report of the death of three children in a car held in a hold-up on a flooded road in which the exhaust was submerged. This study is a toxicological investigation and autopsy report of CO poisoning incidence in four male occupants in traffic jam. We suggest that the government be more responsive in removing heavy broken down trucks that could create traffic jams, build more good and accessible roads to ensure free flow of traffic; close

potholes and remove gallops that could break exhaust pipes producing fuming vehicles. Health education of the public on sources and dangers of CO can also help them stem the increasing reports of CO poisoning in Nigeria.

LIMITATIONS

The specific occupation/ ages were not known to us as we are directly working with the investigators. We are not aware of their occupation as to whether they were working or living in an industrial area.

REFERENCES

1. Prockop LD, Chichkova RI. Carbon monoxide intoxication: an updated review. *Journal of the neurological sciences*. 2007;262(1):122-30.
2. Nnoli MA. Family death-carbon monoxide poisoning from Generator exhaust. *J Medicine in the Tropics*. 2009;11(1).
3. Katsumata Y, Aoki M, Sato K, Suzuki O, Oya M, Yada S. A simple spectrophotometry determination of carboxyhemoglobin in blood. *J Forensic Sci*. 1982;27(4):928-34.
4. Vossberg B, Skolnick J. The Role of Catalytic Converters in Automobile Carbon Monoxide Poisoning A Case Report. *CHEST Journal*. 1999;115(2):580-1.
5. Weaver LK. Carbon monoxide poisoning. *Critical care clinics*. 1999;15(2):297-317.
6. Hampson NB, Zmaeff JL. Carbon monoxide poisoning from portable electric generators. *American journal of preventive medicine*. 2005;28(1):123-5.
7. Sedda AF, Rossi G. Death scene evaluation in a case of fatal accidental carbon monoxide toxicity. *Forensic science international*. 2006;164(2):164-7.
8. Téllez J, Rodríguez A. Carbon monoxide contamination: an environmental health problem. *Revista de Salud Pública*. 2006;8(1):108-17.
9. Horner JM. Anthropogenic emissions of carbon monoxide. *Reviews on Environmental Health*. 2000;15(3):289-98.
10. Kales SN. CO intoxication. *Am Fam Physician* 1993;48(6):1100-4.