Original Research Article

Carbon Monoxide (CO) Pollution in the Niger Delta area of Nigeria and Its Impact on Foeto-Maternal Health

Mkpe Abbey[1], Oloyede O. Adebari[2], Kinikanwo I. Green[3*], Bruno C. Chinko[4]

[1]Department of Obstetrics and Gynaecology, University of Port Harcourt Teaching Hospital, East-West Road, Port Harcourt, Nigeria
[2]Department of Obstetrics and Gynaecology, Olabisi Onabanjo University Teaching Hospital, Hospital Road, 121102, Sagamu, Nigeria
[3]Department of Obstetrics and Gynaecology, University of Port Harcourt Teaching Hospital, East-West Road, Port Harcourt, Nigeria
[4]Department of Human Physiology, University of Port Harcourt Teaching Hospital, East-West Road, Port Harcourt, Nigeria

*Corresponding author: Kinikanwo I. Green
Department of Obstetrics and Gynaecology, University of Port Harcourt Teaching Hospital, East-West Road, Port Harcourt, Nigeria

Abstract

Background: Carbon monoxide is produced in abundance in the Niger Delta, a fact that was acknowledged in the World Bank study of the region in 1995 and the Environmental assessment of Ogoni land by UNEP in 2011. *Objectives:* To ascertain the extent of CO pollution in the Niger Delta, its impact on foeto-maternal health and to review the pathophysiology and the treatment of the poisoning. *Methods:* A mixed method study (observational-descriptive and systemic review). A literature review on the above objectives was carried out. The known foeto-maternal impact of CO exposure was extrapolated to the prevailing state of CO pollution in the Niger Delta. Questionnaires distributed to Doctors working in the tertiary Centres in the core Niger Delta focused on the prevalence, clinical presentations and foeto-maternal findings in patients who presented with CO poisoning. *Results:* There was no data on the prevalence and clinical presentations of CO pollution in the Niger Delta. The ambient and indoors air concentration of CO in the Delta range from 0 ppm to 191µg/m3 but in places within 60-200 metres from crude oil flow stations, the concentrations range from 100 to 5320 µg/m3. Maternal impact of CO pollution ranges from headaches at carboxyhaemoglobin (COHb) levels of 5-20% to maternal death at COHb levels of >66% while in the foetus, it causes birth defects, growth restriction, prematurity and sudden intrauterine and early neonatal death. *Conclusion:* The core Niger Delta is under perpetual siege of CO pollution and the ambient and indoors air concentration of it in the Delta range from 0 ppm to 191µg/m3.
Keywords: Carbon monoxide, Pollution, Niger Delta, Nigeria, Impact, foeto-maternal health.

INTRODUCTION

Carbon monoxide (CO) is a colourless, odourless gas produced by incomplete combustion of hydrocarbons. It enters the body primarily through inhalation, though there is also nominal endogenous production of the gas. The Niger Delta is in the far Southern part of Nigeria where all the petroleum exploration and production take place. Industries are therefore attracted to the region because of the presence of cheap and easy access to oil and gas. Artisanal crude oil refining, oil and gas facilities vandalism, unchecked activities of some multinational companies in the Delta and many other domestic and industrial sources of environmental pollution have earned the Niger Delta a name – ''a region of environmental degradation'' [1].

CO, other gases and teratogens are produced in abundance in the Niger Delta. This widely acknowledged fact has led to series of studies and publications, most notable of which are the World Bank study of the region [2] and the Environmental Assessment of Ogoniland (one of the many ethnic Niger Delta settlements) by UNEP which delivered a catalogue of devastation due to oil pollution in the region. [1] Exposure to CO in pregnancy may be acute or chronic and the foetus has a particular susceptibility to it. It is important therefore for the obstetrician to suspect carbon monoxide poisoning and to know the course of action to be taken when acute or chronic intoxication with the gas in a pregnant woman is encountered.

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Aim

The primary aim of the study therefore is to ascertain the extent of carbon monoxide pollution in the Niger Delta through determination of its indoors and ambient concentration in different regions of the Delta and also determine its impact on foeto-maternal health. The secondary goals of the study are as follows:

a. To review the pathophysiology of carbon monoxide poisoning
b. To review its general presentations and available management options.

METHODOLOGY

The study was carried out at the University of Port Harcourt Teaching Hospital, Rivers State which is situated in the South-South region of Nigeria called the core Niger Delta. It was a mixed method study – observational descriptive and systematic review.

We reviewed published literatures on the impact of carbon monoxide contamination in the Niger Delta on reproductive health. Literature search was carried out, using Pub med (MEDLINE), Biomed central, Google and Cochrane database. Keyword search statements used were: Environmental pollution in the Niger Delta, Sources of CO pollution in the Niger Delta, Gas flaring in the Niger Delta, Petroleum exploration, extraction, refining and CO production, Nigerian norms for CO pollution, WHO norms for CO ambient and indoors air concentration, Physiology of CO poisoning, Effects of CO on adults, Impact of CO on the fetus and mother, Impact of CO pollution on reproductive functions, and Nigerian national register of CO poisoning.

Only studies reported in English language within the last 20 years were included because of the rapid development in the science of environmental studies for the period. Studies carried out in other parts of the world were also included, especially if the studied region has similar environmental terrain like the Niger Delta. We extrapolated the known maternal and fetal impact of CO exposure to the prevailing state of CO pollution in the Niger Delta. The literatures were synthesized by two researchers and relevant information was retrieved from them and agreement on inclusive data was reached by dialogue.

The extent of carbon monoxide pollution in the Niger Delta was assessed by reviewing published data on its concentrations in indoors and ambient air in different regions of the Delta and indirectly by the amount of CO produced by its sources. The identified values were compared with the Nigerian and WHO acceptable ambient and indoor CO concentrations. Telephone communications with the Doctors working in the tertiary health institutions and in the core Niger Delta (Rivers and Bayelsa States) and questionnaires distributed to them focused on the prevalence, clinical presentations and foeto-maternal findings in patients who presented with CO poisoning.

Ethical approval

Ethical approval for this study was granted by the University of Port Harcourt Teaching Hospital Ethical Committee.

RESULTS

Review of articles

Number of articles identified through search = 120. Number of articles dropped for duplication = 10. Number of articles screened = 110. Number of articles that did not meet the inclusion criteria and therefore dropped = 58. Number of articles that met the inclusion criteria = 52. The number of articles actually included in the reference = 44.

The Specialists in obstetrics and gynaecology, internal medicine and accidents and emergency departments of the core Niger Delta states of Rivers and Bayelsa States have not recorded any incidence of CO poisoning for the past 10 years. There was also no data on the prevalence and pattern of clinical presentations of CO poisoning in the Niger Delta. There was no published work on the subject.

The extent of CO pollution in the Niger Delta

CO is produced by the body (endogenous production) and also by human activities (exogenous production). Endogenous production of carbon monoxide results from degradation of blood pigments, namely myoglobin and cytochrome (25%) and heme (75%) [3].

Table 1 illustrates the extent of CO pollution in the Niger delta. In 1995, the total CO emission from Nigeria was 21.42Tg/yr CO, with gas flaring in the Niger Delta being the third main contributor after combustion of biofuels and agriculture. Gas flaring contributed estimated 2.49Tg/yr CO representing 12% of total CO emissions [4]. The Nigerian natural gas that is associated with the extracted crude oil is either flared or vented into the atmosphere [5]. Nigeria is presently the leading gas flaring country in the world, with around 24 billion cubic meters of gas (45% of total production) flared in 2004 [6].

Table 1: Some sources of CO poisoning in the Niger Delta

Authors	Source of exposure	Ambient or indoor air concentration of CO	Life-stage of exposure
Fagbeja, M.A *et al*., 2013 [7]	Domestic sources - Generators firewood and kerosene	70 kt/Year	General population
Jerome, A. 2000 [8]	Road traffic	Lagos 10-250 ppm PH = 5.0-61.0 ppm Warri = 1.0-52 ppm	General population
World Bank (2008) [6]	Nigeria is the leading gas flaring country in the world	Around 24 billion cubic meters of gas (45%) of its total gas production) flared in 2004	Boasts ambient CO air in the Niger Delta.
Oladimeji, 2015a [9]	Gas flaring from Nigerian refineries	Tons of CO / year: Kaduna -56,738.56, Port Harcourt phase 1 - 12,973.20, Port Harcourt phase 2 - 104,230.14, Warri - 67,117.96	General population
Global Emission [4]	Gas flaring	2.49Tg/yr CO representing 12% of total CO emissions in Nigeria in 1995.	General population
Precious N. Ede, David O. Edokpa [10]	Smoking vehicles on Niger Delta roads	Ambient CO μg/m3: Mbiama = 191; >150 in Bonny, Brass, Nchia. Port Harcourt, Sapele and Ughele; >100 in Omoku, Owasa, Eket, Buguma, Ahoada and 100 μg/m3 in Warri and Odukpani. Emuohia and Ukwugba - > 50 μg/m3.	General population Including market women.

Six flow stations situated in the Niger-Delta were monitored daily for four consecutive months for concentration of CO at certain distances from the stations [11]. The result was as shown in Table 2.

Table 2: Concentration of CO around the Flow Stations [11]

Flow Station	Concentration ($\mu g/m^3$)					
	Distance (Morning Sampling Period)			Distance (Afternoon Sampling Period)		
	60 m	200 m	500 m	60 m	200 m	500 m
1	300-2350 (1800±1000)	750-850 (800±50)	450-2750 (1100±1130)	400-3750 (2800±1580)	400-3750 (2800±1580)	500-3250 (1200±1380
2	2100-2350 (2300±130)	750-850 (800±50)	500-550 (500±30)	3550-3850 (3700±130)	2450-2650 (2600±90)	500
3	980-5350 (3400±2180)	750-1370 (1190±300)	100-900 (500±450)	1150-2350 (1700±700)	190-1960 (1150±750)	100-1140 (400±490)
4	630-1740 (1200±610)	300-3850 (2800±1680)	400-750 (400±250)	750-2200 (1700±660)	900-6530 (4600±2650)	450-1100 (500±290)
5	2300-2350 (2500±38)	700-1100 (900±220)	100-2360 (1300±1120)	100-2140 (900±890)	750-1100 (1000±180)	100-850 (500±310)
6	100-2710 (800±1270)	100-430 (250±140)	100-1710 (500±540)	100-2550 (700±1230)	100-170 (140±30)	100-1850 (500±880)

Air quality was checked in 3 slums (Maroko, Igbudu and Marcaver) located by River banks and on pipeline right of way in Warri [12]. Warri is located in the core Niger Delta in Southern part of Delta State, Nigeria. It is a major urban city where many sources of air pollution are located. Air quality for carbon monoxide (CO) and other gases was monitored in the living room and kitchen areas of 20 households in each of the chosen slums, i.e. 60 households [12].

In Maroko, Makaver and Igbudu, the average concentrations of CO (ppm) in living room (Pallor) and kitchen were significantly different from each other.

They were 28.70 (21-37) and 34.20 (27-41), 19.05 (12-31) and 23.85 (21-34) and 33.0 (26-39) and 38.55 (29-46) in living room and kitchen respectively. The obtained values for CO in the study may have resulted from the form of domestic fuels used for cooking, namely firewood, wood charcoal and sawdust. Biomass and coal smoke have been reported to emit many health-damaging pollutants, including particulate matter (PM), carbon monoxide (CO), sulphur oxides, nitrogen oxides, aldehydes, benzene, and polyaromatic compounds [13].

A study on the ambient air concentrations of CO in different areas of Port Harcourt, a city in the core Niger Delta and its surrounding, showed various levels of CO contamination as enumerated in table 3 below [14].

In 2015, CO levels was checked in some towns in the Niger Delta, namely Ahoada, Bonny, Brass, Buguma, Ukwugba, Eket, Nchia, Emuoha, Mbiama, Odukpani, Omoku, Owasa, Port Harcourt, Sapele, Ughelli and Warri [10]. These settlements straddle six states in the region as well as the ecological and climatological sub-divisions of the Niger Delta. CO concentrations were highest in Mbiama (191 μg/m3) [10], a small town used by long distant trailer drivers as a stopover place in their journey to different parts of Nigeria. CO concentration was above 150 μg/m3 in Bonny, Brass, Nchia, Port Harcourt, Sapele and Ughele; above 100 μg/m3 in Omoku, Owasa, Eket, Buguma, Ahoada and 100 μg/m3 in Warri and Odukpani [10].

Table 3: Ambient air concentrations of CO in different areas of PH

Serial Number	Area	Category of the area	Ambient CO (ppm\)
1	Port Hacourt Airport, Omagwa	Business	51
2	Rumuokoro Junction	Commercial/ Highway Traffic	53
3	Agip Petroleum	Industrial	36
4	Mile 3 Market Diobu	Commercial	35
5	Ikoku Spare Part Market, Olu Obansjo.	Commercial / Traffic	35
6	Mummy-B Rd, GRA	Residential	36
7	Rumuola Bridge	Commercial/ Traffic	34
8	Eleme Petrochemical	Industrial	37
9	Eleme Junction, Onne Rd	Mixed/Traffic	36
10	Onne Wharf	Business	35
11	Eleme Refinery	Industrial	35
12	Elele Alimiri Village	Commercial / Traffic	37
13	Elele junction	Commercial/ Traffic	35

Emuohia and Ukwugba had the least concentration of CO but > 50 μg/m3. All CO measurement in the study exceeded the WHO limits and it may be due to the prevalence of too many smoking vehicles on Niger Delta Nigeria roads.

Indoors and ambient concentration of CO in the Universities in the Delta was illustrated with the findings at the UNIPORT in the core Niger Delta [15]. The concentrations ranged from 0-192 ppp. In most of the offices, 0 ppp was recorded. High levels were recorded in the business park and in some of the offices, probably due to the many generators that were used for electricity.

The Pathophysiology of CO poisoning
Carbon monoxide is inhaled from air, dissolves in plasma and binds to the oxygen transporting proteins, namely haemoglobin (Hb) in the plasma and myoglobin and the cytochrome system within tissues, thereby perturbing oxygen exchange in the tissues. It binds to HB rapidly and avidly with an affinity more than 200 times that of oxygen [16-18] and by a stable and reversible reaction - Oxyhemoglobin (HbO$_2$) + CO = Carboxyhaemoglobin (COHb) + 0$_2$. It therefore causes a leftward shift in the oxygen–haemoglobin dissociation curve, decreasing oxygen delivery to tissues, resulting in tissue hypoxia. [18] The amount of COHb formed depends on the duration of exposure to CO, the concentration of CO in the inspired air and the alveolar ventilation.

CO also binds to intracellular myoglobin and cytochromes (P450) [19]. It also binds with guanylyl cyclase. These processes create a reservoir and accounts for the persistence of carbon monoxide poisoning, even with oxygen therapy. [20] CO causes hypoxia/ischemia due to COHb formation and direct CO toxicity at the cellular level. This combination helps to explain why COHb levels do not correlate with the severity of clinical effects [18-27].

Carbon monoxide dissolved in maternal plasma crosses the placental barrier by passive diffusion. Facilitated diffusion of CO through the placental membrane has also been suggested [28, 29]. Foetal carboxyhemoglobin (COHbF) levels are 0.7-2.5%. The concentration of carboxyhaemoglobin in the mother is slightly lower than in the foetus, with virtually no difference in non-smokers, but with a significant difference in smokers [21]. Two key mechanisms are responsible for the toxicity of carbon monoxide in the foetus, namely tissue hypoxia, and direct action of CO on the body's haem-containing proteins, such as myoglobin and the cytochromes. Foetal haemoglobin affinity for CO is 172 times greater than that for oxygen.

Kinetics of Carbon Monoxide
The elimination of carbon monoxide occurs because the higher oxygen levels in the lung favour the dissociation of carboxyhaemoglobin, allowing free carbon monoxide to be exhaled at a rate dependent on the partial pressures of the two gases. For brief acute

poisoning the estimated half-life of carbon monoxide in the human is about 4 hours when breathing in ambient air, 1 hour 30 minutes in pure oxygen at atmospheric pressure, and 20 minutes in hyperbaric oxygen at 3 atmospheres.

During the first two or three hours of chronic carbon monoxide poisoning maternal COHb concentration rises rapidly and then slowly, reaching a plateau in 7 to 8 hours. COHbF reaches the maternal levels in 14 to 24 hours in female humans. A state of equilibrium is reached in 36 to 48 hours, with the concentration COHbF being 15-20% greater than maternal COHb. The elimination half-life is 2 hours for the mother and 7 hours for the foetus, and therefore the duration of hyperbaric oxygen therapy needs to be longer for pregnant women than for women who are not pregnant.

F0eto-maternal impact of CO exposure

In acute poisoning, fatal outcome is proportional to the severity of maternal involvement and is associated with carboxyhaemoglobin levels in the mother [30]. This is not true during chronic poisoning where the foetus can be much more severely affected than the mother. Presentation in the mother is as follows: chronic poisoning with maternal COHb levels of 5-20% - headaches, impaired physical performance, sensations of weakness, dizziness, sleepiness, visual difficulties, palpitations, nausea, vomiting; acute poisoning at COHb levels of 30-50% - tachypnoea, respiratory insufficiency, tachycardia, hypotension, arrhythmia fever, confusion, disorientation, alteration in consciousness, convulsions, vomiting; life-threatening poisoning at C0Hb levels of 50-66% - psychiatric difficulties, alteration in consciousness, apathy, apraxia, disorientation, muscular hypertonia, intestinal problems, urinary or faecal incontinence and lethal poisoning at COHb levels of >66% [31]. The COHb levels in percentages represent the percentage of displaced oxygen by CO from carboxyhaemoglobin,

The toxic effects of carbon monoxide vary according to the gestation at which the poisoning occurs. Chronic exposure to CO during the first two trimesters of pregnancy can produce significant intrauterine growth restriction, presumably due to chronic hypoxia. CO poisoning potentiates oxygen deficiency, and intrauterine growth restriction can be very severe. Other toxic effects include preterm labour [32], intrauterine foetal death [33], and sudden infant death [34]. Foetal death can occur in the absence of severe maternal symptoms. During acute severe intoxication the foetus may die of anoxia because maternal haemoglobin is saturated with carbon monoxide. The death by anoxia occurs well before the levels of fatal carboxyhaemoglobin have had time to rise [35] foetal CO poisoning can be diagnosed retrospectively by measuring fatal carboxyhaemoglobin

post-mortem which remains unchanged for several days.

Maternal exposure to CO during organogenesis is associated with formation of significant congenital abnormalities. They are as follows: Neurological defects - telencephalic dysgenesis (heterotopia, pachygyria, schisencephalia) [36], behavioural difficulties during infancy [37], skeletal effects namely hand and foot malformations, hip dysplasia and sublaxation, agenesis of a limb, inferior maxillary atresia with glossoptosis [38]; and Cleft palate. Maternal exposure during the fatal period is associated with anoxic encephalopathy [39], while in the 3rd month of pregnancy, cardiac defects, including ventricular septal defects (VSD), right-sided cardiomegaly (caused by myosites hyperplasia) may occur.

Investigations

Recommended investigations in women with suspected carbon monoxide poisoning are as follows: COHb which gives an indication of the severity of maternal poisoning and a cruder indication of the degree of the poisoning [40], blood gas [41], toxic screen to detect any other poison, serum creatinine phosphokinase and low-density lipoprotein levels [42], liver function test, serum glucose level as secondary hyperglycaemia can sometimes be observed, full blood count to look for leucocytosis, electrocardiogram since myocardial ischaemia or cardiac arrhythmia may occur and neuropsychological testing as discrete abnormalities of higher cortical function may be a sequel. CT or MRI of the head to identify cerebral oedema or infarction and EEG to identify diffuse abnormalities of cerebral electrical activity.

Smokerlyzer

A hand-held instrument called Smokerlyzer has been used to measure the concentration of CO in expired air especially in smokers. It displays CO ppm; the %COHb and %FCOHb are calculations based on clinical evidence. COHb = 0.63 + 0.16(EC50), where (EC50) is the concentration of CO in ppm that is expired after inhalation as measured with a Smokerlyzer [43].

Treatment

If signs of acute poisoning are present or the COHb is greater than 20%, hyperbaric oxygen therapy should be administered as soon as possible. Immediate delivery carries a high risk of perinatal death in the period of acute poisoning, and so hyperbaric oxygen must always be given first before performing an emergency caesarean section. After the acute stage has passed, prediction of fatal outcome is difficult. Ultrasound and magnetic resonance imaging of the fatal brain are the most useful investigations for the detection of anoxic cerebral necrosis. It is often only following

birth that a realistic evaluation of the future consequences to the infant can be made.

Hyperbaric oxygen therapy increases the dissolved oxygen concentration, accelerates the dissociation of COHb and reverses the binding of CO to myoglobin and the cytochrome system, shifts the oxyhemoglobin dissociation curve to the right, which favours the liberation of oxygen into the tissues. Ultimately CO is eliminated from the mother and the foetus and fatal hypoxia is reduced [44]. A 100% oxygen therapy should be administered while waiting for hyperbaric oxygen after the acute episode has been treated. Long term fatal cardiac monitoring should be performed. Other treatment modalities are based on the prevailing symptoms and signs.

DISCUSSION

The study showed that there was no case of CO poisoning in the Niger Delta, but we cannot rule it out completely; it may be misdiagnosed as other conditions. The pockets of studies that were reviewed in the study showed that the Delta was under profound siege of CO pollution. Nigeria is the leading gas flaring country in the world and almost all the gas flaring which produces high concentrations of CO occurs in the Niger Delta.

Three of the four Nigerian refineries which contribute immensely to CO pollution are located in the Niger Delta. They are Warri refinery, Port Harcourt (PH) Refinery phase 1 and phase 2 [7]. Worse still, the PH refineries 1 and 2 are confined to a village called Alesa in Rivers State in the Niger Delta. It means, the inhabitants of the village and its surrounding have been exposed to high concentrations of CO for several years.

Inhabitants of the Niger Delta live on the right of ways of oil pipes and very close to flow stations which are built all over the region. The nearer the flow stations to the dwelling places the more the concentrations of ambient CO. Unfortunately, people live as near as 500 metres to the station, thereby exposed to high doses of it [11]. The problem is further compounded by smoking vehicles which ply on Niger Delta roads.

Ambient CO concentrations in the region ranged from 100-191 μg/m3, with few towns having 50-100 μg/m3 [9]. Inhabitants of the Delta are also exposed to high indoor concentrations of CO because firewood and kerosene are used as biofuel for cooking and generators as source of light. Levels of 19.05- 33.0 ppp in living rooms and 23.85 – 38.55 ppp in kitchens have been recorded [12]. In offices, e.g. in the University of Port Harcourt, CO concentrations ranged from 0-192 ppp due primarily to the use of generators for light. Generally, the figures for indoor CO concentrations were far more than that of the WHO.

Much was learnt about the pathogenesis, presentations, investigations and treatment of CO poisoning. The high ambient and indoor concentrations of CO in the Niger Delta showed that acute and chronic poisoning with the gas is possible in pregnancy. In view of the non-specific presentation of chronic CO poisoning, the diagnosis is likely to be missed. Restricted fatal growth, preterm labour and sudden infant death can also occur. All these imply that as soon as the mother attends with clinical signs of intoxication, and especially when the carboxy-haemoglobin concentration is > 20%, hyperbaric oxygen therapy should be started urgently. 100% O_2 should be given where the hyperbaric oxygenation is not available.

RECOMMENDATIONS

Firstly, the full WHO guideline on CO should be adopted so that accurate and unified comparison can be achieved in the Niger Delta and Nigeria at large. Secondly, there should be high index of suspicion when a Maternity presents with symptoms and signs of possible CO toxicity. A register of poisoning should be maintained in the Nigeria Delta so that the true burden of the problem can be ascertained. Furthermore, there is need for unified Niger Delta ambient air quality assessment as practised in developed countries. This will go a long way identifying those regions that are worse affected, preventive measures can be instituted appropriately and the-already stressed medical services in the Delta and Nigeria at large can be triaged appropriately.

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