

Case report:

Accidental cooking gas intoxication

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Received: 23rd Apr 2008; Accepted: 4th Jan 2009.**ABSTRACT**

The primary component of natural gas is methane (CH₄). It also contains ethane (C₂H₆), propane (C₃H₈), butane (C₄H₁₀), and other sulfur containing gases, in varying concentrations.

Suicide by domestic gas was forming more than 40 percent of the annual number of suicides in England and Wales in 1963.

Jarvis *et al.* reported that women using gas stoves had double the respiratory problems of women cooking on electric stoves. I am reporting three cases of accidental cooking gas intoxication, with history of unconsciousness, with or without convulsion. The two males among our patients presented were ended up with neurological deficits like abnormal movements, disorientation and irritability. The 3rd patient recovered more or less completely but still she was complaining from mild weakness in the lower limbs. Natural gas carries an important cause of respiratory and neurological illnesses if the patients are exposed to it for enough time.

الخلاصة

إنّ المكونات الأساسية للغاز الطبيعي هي: الميثان بصورة أساسية وكذلك يحتوي على الإيثان، البروبان، البيوتان، وغازات تحتوي على الكبريت. وكمية هذه الغازات بنسب متفاوتة..

الانتحار بالغاز الطبيعي كان يشكل أكثر من ٤٠ بالمائة من العدد السنوي لحالات الانتحار في إنجلترا وويلز في ١٩٦٣. جار فيس وجماعته توصلوا إلى أن النساء اللواتي يستعملن الطباخات الغازية كان عندهن نسبة الإصابة بالأفات الرئوية كالربو ضعف ما كان يصيب النساء اللواتي يستخدمن الطباخات الكهربائية.

أردت تسجيل ثلاثة حالات تسمّم عرضي بالغاز المستخدم في الطبخ: ثلاثة مرضى أحيلوا إلى وحدة العناية المركزة والتنفسية في مستشفى ابن سينا التعليمي بالموصل، رجلان وامرأة وكانوا جميعا في حالة فقدان الوعي، مع الإصابة بنوبات صرع متكرر. اثنان من مرضانا انتهوا بخلل في الجهاز العصبي المركزي مثل الحركات غير الإرادية في الجذع والأطراف مع تشوش الذاكرة خصوصا في تحديد المكان، أما المريض الثالث (الأنثى) فقد تعافت تقريبا بالكامل ولكنها بقيت تشتكي من ضعف بسيط في الأطراف.

الغاز الطبيعي مسبب رئيسي للأمراض التنفسية والعصبية إذا تعرّض له المرضى واستنشقوا الغاز لفترة طويلة مناسبة.

The primary component of natural gas is methane (CH₄); the shortest and lightest hydrocarbon molecule. It also contains heavier gaseous hydrocarbons such as ethane (C₂H₆), propane (C₃H₈), and butane (C₄H₁₀), as well as other sulfur containing gases, in varying amounts as in (Table 1)⁽¹⁾.

Suicide by domestic gas was accounting for more than 40 percent of the annual number of

suicides in England and Wales in 1963. At the year 1975 this number showed a sudden, unexpected decline from 5,714 to 3,693 at a time when suicide continued to increase in most other European countries. This appears to be the result of the progressive removal of carbon monoxide from the public gas supply⁽¹⁾.

In 1996 Jarvis *et al.* reported that, in the UK, females who used gas cookers had an increased risk of wheeze and other asthma symptoms, as well as lower lung function (FEV₁ and FEV₁/FVC) than females not using gas cookers.⁽²⁾

Furthermore they found that women using gas stoves had double the respiratory problems of women cooking on electric stoves. When natural gas is burned, the chemicals byproducts create nitrogen dioxide, carbon monoxide, fine particulates, polycyclic aromatic hydrocarbons, volatile organic compounds (including formaldehyde) and other compounds⁽²⁾. Among the top10 leading sources of fatal work-related inhalations in the United State of America are *coal, natural gas, and petroleum fuels* and its products; they form about (11.8%) of the total number of dead patients⁽³⁾. Butane is a derivative of natural gas which is used in gas lighter refills; its abuse by inhalation of cigarette lighter refills and aerosol propellants can cause many health hazards⁽⁴⁾.

I am reporting three cases of accidental cooking gas intoxication referred to the Respiratory and Intensive care Unit (RICU) at Ibn-Sena Teaching Hospital in Mosul during the cold weather period from November 2004 to early January 2005. The three patients had a history of collapse while they were in the bathroom, discovered by their families 30-45 minutes after their entrance to the bathroom. Their families managed to open the door of the bathroom by force after 30-40 minutes where they found them unconscious, with or without convulsion and there was a leaking cooking gas heater (locally made) that were used by the patients to warm the bathroom and the water for bathing.

Case report (1): A 23 -year- old male patient referred to the RICU at Ibn-Sena Teaching Hospital from the casualty department on the 30th of November 2004. The patient was brought to the casualty department where he developed a convulsion and then referred to the RICU. The on arrival examination details in the RICU are as in (Table 2).

The various laboratory investigations as in (Table 3). The CT scan brain and MRI brain

are useful investigation for unconscious patient with convulsion, but the CT scan was not working in our hospital at that time and the MRI brain was impossible to be done for our patients because the patients were not cooperative and the anesthetist in the MRI unit refuses to give adult patients anesthesia as they use anesthesia for children only and this applies for the other two patients.

The immediate physical signs were similar to increased intracranial pressure, so we started him on dexamethazone 4mg 6 hourly, mannitol 200mL during 30 minutes twice daily and ampicillin-cloxacillin 500mg IV 6hourly. The patient was given high oxygen concentration and to be mechanically ventilated if the SpO₂ remain low.

The patient was monitored hourly as usual in the RICU with continuous SpO₂ monitoring.

Arterial blood gas analysis was not available. On the 2nd of December the patient became conscious but irritable with slurred speech and abnormal chorioathetosis-like movements of his upper and lower limbs, slightly disoriented for place. The respiration was normal and his SpO₂ on room air was 98%. Neurological consultation confirmed the diagnosis of chorioathetosis, and the patient neurological abnormality was treated accordingly.

The patient continues to be slightly disoriented for place with the same abnormal movement but it was less than before even two months later when he was seen for follow-up.

Case (2): A 24 years male patient referred to the RICU at Ibn-Sena Teaching Hospital from the casualty department on the 26th of December 2004. The patient was brought to the casualty department and then referred to the RICU. The on arrival examination details in the RICU are as in (Table 2). The laboratory investigations as in (Table 3).

The immediate physical signs and treatments of the patient were similar to case number one. The patient monitored hourly as usual in the RICU with continuous SpO₂ monitoring.

On the 27th of December the patient regained his consciousness although he was disoriented for place with slurred speech; he became irritable with abnormal

chorioathetosis-like movements of the trunk and the extremities. The patient was treated accordingly. The SpO₂ became 98% with normal respiration.

The patient was brought for follow up three weeks later with the same disorientation for place, irritability and mild abnormal chorioathetosis-like movements.

Case (3): An 18-year-old female patient referred from Telafar Hospital directly to the Respiratory and intensive care unit at Ibn-Sena Teaching Hospital on the 23rd of January 2005. The on arrival examination details in the RICU were as in (Table 2). The immediate treatments of the patient were similar to the first and second patients. The ordinary laboratory investigations as in (Table 3). The patient monitored hourly as usual in the RICU with continuous SpO₂ monitoring.

On the 24th of January 2005 the patient was fully conscious but unable to walk.

Neurologically there was generalized weakness in both lower limbs; all reflexes were intact and the plantar reflex was flexor with no localizing sign .

By the 26th of January the patient was able to walk for a short distance alone and she felt weak in both lower limbs and she was happy to be discharged home. The patient failed to come for follow up.

Table (1): The Components of Natural Gas

Component	wt. %
Methane (CH ₄)	70-90
Ethane (C ₂ H ₆)	5-15
Propane (C ₃ H ₈) and Butane (C ₄ H ₁₀)	< 5
CO ₂ , N ₂ , H ₂ S, etc.	balance

Table (2): The physical signs and symptoms during examination in the RICU

Symptoms and Signs	Case 1	Case 2	Case 3
Pulse	Tachycardia	Tachycardia	Tachycardia
SpO ₂	58%	62%	70%
Blood Pressure	Normal	Normal	Normal
Breathing	Tachypnoea and Periodic	Tachypnoea	Tachypnoea
Cyanosis	Present	Present	Mild Cyanosis
Conscious Level (GLASGOW COMA SCALE)	GCS=5	GCS=6	GCS=10
History of Convulsion	Present	Present	Present
Pupils	Normal in size and reacting to light	Normal in size and reacting to light	Normal in size and reacting to light
Plantars	Equivocal	Normal	Normal
Auscultation of the Chest	Diffuse crackles bilaterally	Harsh Vesicular Breathing with scattered crackles	Harsh Vesicular Breathing
Chest x-ray	Increased lung markings	Normal	Normal
ECG	Sinus Tachycardia	Sinus Tachycardia	Sinus Tachycardia

Table (3): The investigations done for the three patients

Cases	Hb g/L	WBC	FBS or random mmol/L	B.UREA mmol/L	S.CREATININE μmol/L	S.Na. mmol/L	S.K mmol/L
Case-1	139	8.6	R(9.2)	5.5	109	140	4.4
Case-2	90	9.7	6.4	5.0	100	135	4.0
Case-3	120	7	6.0	7.0	80	136	3.5

Discussion

Natural gas brings harmful chemicals into homes through the methane and other components it contains. Methane (which gives the flame its blue colour as it does in propane) is an asphyxiant chemical.

The natural gas typically contains radon and other radioactive materials, BTEX (Benzene, Toluene, Ethylbenzene and Xylene), organometallic compounds such as methylmercury, organoarsenic and organolead. Mercaptan, an odorant, is also added to natural gas so that it can be detected by smell before reaching the toxic or explosive levels⁽⁵⁾.

The patients presented in this paper were intoxicated accidentally by inhalation of cooking gas rather than due to abuse. The intoxication developed due to the use of the only available cooking gas stoves for heating the bathrooms and to have hot water for bathing.

The intoxication with asphyxiant gases usually started clinically as euphoria, excitation, blurred vision, slurred speech, nausea, vomiting, coughing, sneezing and increased salivation. If the intoxication continues then disinhibition, confusion, perceptual distortion, hallucinations, delusions, tinnitus and ataxia develop. If the dose increased, the patient will start to have nystagmus, dysarthria, tachycardia, central nervous system depression, drowsiness, coma and sudden death which may result from anoxia, vagal inhibition of the heart, respiratory depression, cardiac arrhythmias^(6,7).

There is no typical clinical finding of inhalation poisoning apart from possible unconsciousness, the diagnosis is usually

made depending on the history of exposure to the gas in badly ventilated space^(4,8).

The three patients I am presenting were unconscious at presentation and it was impossible to take history from them and they were in severe hypoxic state as they were centrally cyanosed with their SpO₂ 58%, 62% and 70% (case 1, 2 and 3 respectively). Furthermore all the three patients presented with convulsion as an indication of cerebral anoxia due to the long period of intoxication which the three patients sustained and these findings are in accord with previously mentioned studies^(6,7).

Patients with loss of consciousness are at high risk of developing delayed neuropsychiatric symptoms, which vary from mild intellectual impairment or personality changes to specific neurological deficits such as deafness, blindness, and Parkinsonism⁽⁹⁾. The previously mentioned neurological deficits occurred in our case number one, case number two and to a lesser extent in case number three.

In the United Kingdom during 1988-1990, 398 people mainly teenagers died due to abuse of fuel gas. Butane (as a derivative of natural gas) inhaled from lighter refill canisters has accounted for three times as many deaths as any other abuse of fuel gas products⁽¹⁰⁾.

Gunn *et al.*, 1989 reported a boy aged 15 years with a habitual inhalation of butane by spraying it on his towel and then inhaling it to get euphoria where he developed ventricular fibrillation followed by apnea and he was ventilated for 36 hours due to cerebral oedema and made complete recovery⁽¹¹⁾. Furthermore Bauman *et al.*, 1991 reported a myocardial

infarction in a boy aged 15 years; the boy was found unresponsive and cyanosed after inhaling butane from a plastic bag. The patient developed generalized tonic clonic seizures. The patient's ECG indicates anterolateral myocardial infarction. The patient was ventilated for 13 days, on discharge the patient left with memory and personality problems^(12, 13).

Gray and Lazarus in 1993 reported right-sided hemiparesis in a 15 year old boy after inhalation of a half a can of butane. The patient was left with pronounced proximal muscle weakness of the upper limbs and hemiplegic gait and his CT scan was normal⁽¹⁴⁾.

Adgey *et al* reported in UK, that fuel gases appeared to be responsible for about 30% of deaths due to solvent abuse and aerosol propellants responsible for 20%⁽¹⁵⁾. Furthermore Chaudry in the year 2002 reported 64 deaths from volatile substance misuse, more than 50% of them were attributed to fuel gas inhalation⁽¹⁶⁾.

The patients presented in this paper were intoxicated accidentally by Fuel gas and none of them were suffering from cardiac problem at presentation apart from the sinus tachycardia.

Our three patients were presented with neurological problems like convulsion and loss of consciousness, these findings are in accord with the finding by Doring *et al* as they reported severe encephalopathy in a 15 year-old girl due to abusive butane inhalation⁽¹⁷⁾.

Furthermore two of our patients presented ended up with neurological deficits like abnormal movements, disorientation and irritability; the 3rd patient recovered more or less completely but still she was complaining from mild weakness in the lower limbs. Similar abnormalities were reported in the USA by Bowen *et al* during the period 1987-1996.

They reported 39 cases in Virginia who likely died as a direct consequence of exposure to an abused inhalant with definite CNS effects like behavior changes, slow speech, elated mood, hallucinations and illusionary experiences⁽¹⁸⁾.

It is fair to say that acute carbon monoxide poisoning may cause similar clinical picture,

but we assume that our cooking gas brand was from the new generation i.e. carbon monoxide free cooking gas, due to the progressive removal of carbon monoxide from the public gas supply which was carried out by the Petrol companies starting from the early sixties⁽¹⁾. Furthermore the pulse oximeter measures both carboxyhaemoglobin and oxyhaemoglobin, therefore it may indicate a normal value in carbon monoxide poisoning which were not the case in our patients. The pulse oximeter measure of our patients returned to normal in nearly the 2nd or 3rd day without using the hyperbaric oxygen therapy which is usually required in carbon monoxide poisoning although its use is controversial now a days⁽¹⁹⁾.

Conclusion

Natural gas carries an important cause of respiratory and neurological illnesses if the patients exposed to it for enough time, especially in those who use the open flame gas cook stoves, hot water heaters, and furnaces.

There are good medical evidences indicating clearly that natural gas should be restricted to generating electrical energy; this kind of legislation is now approved in Canada.

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