

Gas Geyser Syndrome: A Case Series

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Abstract: To bring to light the greatly hazardous effects of the use of fuel less gas geysers in the domestic setting. Many cases were documented as presenting with unexplained neurological events while bathing in an ill ventilated bathroom with a functional fuel less gas geyser. To increase awareness regarding gas geyser associated carbon monoxide intoxication and neurological symptoms, both of which are entirely preventable conditions. We also wish to emphasize the importance of stringent and universal implementation of gas geyser usage and installation laws.

Keywords: Carbon monoxide, epilepsy, gas geyser

1. Introduction

Liquefied petroleum gas (LPG) geysers are used very frequently for heating water in developing countries such as India. These geysers are simple, economical in the form of faster heating of water and are used in bathrooms for bathing purpose. The electrical supply is erratic in developing countries; as a result, cheaper modes such as gas geyser for heating water for bathing are utilized. [1] However, these gas geysers emit various toxic gases; one among them is colorless, odorless carbon monoxide (CO). In the past few years, there were reports of unexplained loss of consciousness in the bathroom. Users in most of the cases recovered spontaneously with few having life-threatening after effects. These episodes were attributed to seizures as hot water epilepsy, head injury, stroke, cardiac events, and vasovagal episodes. [2] However, the exact cause for these episodes has been recognized as *toxic encephalopathy* due to toxic gases inhalation, mainly carbon monoxide (CO). The need for awareness of gas geyser syndrome is necessary to prevent fatalities. Here, we describe the patients brought to our Emergency Department with loss of consciousness in the bathroom while bathing over a period of 2 years.

Case 1

A 28-year-old woman had a fall while bathing with hot water. When she did not come out from the bathroom for nearly 1 h, her family members became suspicious and broke open the door. She was found unconscious on the floor. There was no froth from the mouth. She was rushed to hospital where she gradually regained consciousness over the next 1 h. On examination, there was no tongue bite. Her vitals were stable. There was no evidence of any neurological deficit. Her CT brain (FIGURE 1) with MR diffusion brain was normal. Arterial blood gas analysis showed metabolic acidosis. All routine blood tests including serum creatine phosphokinase (CPK) and EEG were normal. She was diagnosed as hot water epilepsy (HWE) and advised to take prophylaxis with clobazam. HWE is a term used to encompass a reflex epileptic condition, characterized by pouring hot water (40–50°C) on the head. [13] although it has been reported from all parts of the world, it is more prevalent in the Southern Indian population as most of the reported cases have been from there. [12] On detailed history regarding usage of gas geyser revealed that they were using gas geyser that was located completely (with burner and cylinder) inside the small-sized bathroom. A

diagnosis of possible CO intoxication was made and was advised to use gas geyser in a well-ventilated bathroom.



Figure 1: Normal CT Brain

Case 2

A 19-year-old girl was found unconscious in the bathroom while bathing. She was taken to local hospital, and an initial diagnosis of seizure was made. She was started on injectable antiepileptic drugs and referred to our hospital for further management. On examination, the heart rate was 130 beats/min; blood pressure was 100/68 mmHg, and respiratory rate was 20/min. There were injury marks on the back but no marks of ligature around the neck. Neurologically, she was deeply comatose with decerebrate posturing to painful stimulus. Pupils were mid-dilated, with sluggish reaction to light. All deep tendon reflexes were sluggish, and both plantar responses were extensor. Complete hemogram, renal, liver, and thyroid function tests, and serum electrolytes were normal. Arterial blood gas analysis showed metabolic acidosis (corrected).

Electrocardiogram revealed sinus tachycardia. Blood screening for toxic substances was negative. Computed tomography of the brain showed loss of gray-white matter differentiation with hyper dense basal ganglia and cerebellum suggestive of hypoxic brain insult [Figure 2].

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EEG showed diffuse slowing of background rhythm suggestive of diffuse brain dysfunction. A provisional diagnosis of toxic/hypoxic encephalopathy was made. She was started on anti-oedema measures with neuro-protective agents. Within 7 days, she succumbed to her illness. It was found that she was the first to take bath that day and had taken longer time for bathing in a gas geyser-fitted bathroom with ill-ventilation. The blood sample was sent for CO level estimation but was negative.

Case 3

An 18-year-old boy had a fall while bathing with hot water. When he did not come out from the bathroom for nearly 1 h, his family members became suspicious and broke open the door. He was found unconscious on the floor. There was no froth from the mouth. He was rushed to hospital where he gradually regained consciousness over the next 1 h. On examination, there was no tongue bite. His vitals were stable. There was no evidence of any neurological deficit.

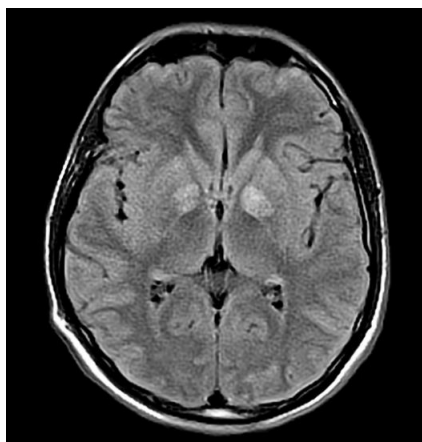


Figure 2: Bilateral globus pallidus T2/F 1

His family members were initially reluctant to go ahead with neuroimaging. Arterial blood gas analysis was normal. On detailed history suggested usage of gas geyser that was located completely inside the small-sized bathroom. A diagnosis of possible CO intoxication was made and was advised to use gas geyser in a well-ventilated bathroom. No confirmatory tests were done.



Figure 3: Normal CT Brain

Case 4

A 21-year-old woman with alleged history of sudden fall in the bathroom while bathing. She was brought to the EMD in an unconscious state. On examination, the heart rate was 140 beats/min; blood pressure was 130/90 mmHg with SpO₂ 60% on room air. There were injury marks on the back but no marks of ligature around the neck. Neurologically, she had a GCS 7-8/15. Patient was intubated in view of poor GCS and taken on ventilator support. Arterial blood gas analysis was s/o high anion gap metabolic acidosis with very high lactate level. On auscultation, patient has bilateral coarse crepitations (left>right). Detailed history suggested post-COVID and post COVID vaccination status. CT brain (FIGURE 3) with MR diffusion was normal. HRCT thorax showed ground glass opacities in left lower lobe and upper lobe with apical and posterior basal segment of right lower lobe. All routine blood tests including serum creatine phosphokinase (CPK) and EEG were normal. Coximetry was done which was confirmatory of carboxy hemoglobin (CO-Hb) of 28%. It was found that she took bath that day and had taken longer time for bathing in a gas geyser-fitted bathroom with ill-ventilation. Patient was started on prophylactic antibiotics and extubated after 24 hours of ventilatory support.

CO-oximeter-Mechanism

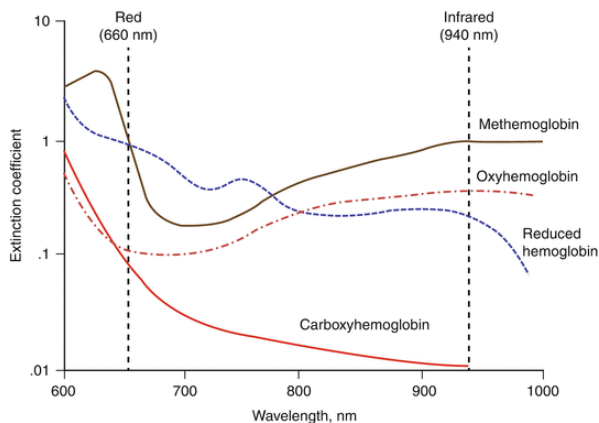
A CO-oximeter (figure 4) measures the absorption of light passing through blood from few as two or three wavelengths of light to several dozens of wavelengths, in order to distinguish oxyhemoglobin, and deoxyhemoglobin (formerly called 'reduced' hemoglobin), and thus determine the oxyhemoglobin saturation (the percentage of oxygenated hemoglobin compared to the total amount of available hemoglobin (Hb)).



Figure 4: CO-oximeter

Measurement of greater numbers of wavelengths enables the instrument to distinguish between these and carboxyhemoglobin, -COHb, methemoglobin-metHb, other hemoglobin moieties and 'background' light-absorbing species. Traditionally, measurement is made from arterial blood processed in a specific device designed to be able to measure proportions of multiple components of several hemoglobin moieties using multi-wavelength

spectrophotometry and complex, but straightforward internal computations. While these units still are in wide use, blood gas analyzers with integral CO-oximetry modules have also been developed and successfully marketed by several manufacturers. More recently, some 'pulse' or more precisely 'peripheral' oximeters have made it possible to estimate carboxyhemoglobin with non-invasive technology similar to a simple (peripheral) pulse oximeter. In contrast, the use of a standard or simple pulse oximeter is not effective in the diagnosis of CO poisoning as patients suffering from carbon monoxide poisoning may have a normal oxygen saturation reading on a pulse oximeter.^[14]



Risk Factors

- Winter season
- Small bathroom
- Ill ventilated/closed windows
- Poor manufacturing (substandard brand)
- Female (taking longer time in bathroom)
- Tandem bathing pattern (one after another family members going for bath)

Pathophysiology (Toxic Gases)

- LPG – butane/propane
- Co₂
- Co
- Nitrous oxide
- Hydrocarbon gases (HC)

Treatment

Prevention is the best treatment.

Initial treatment for carbon monoxide poisoning is to immediately remove the person from the exposure without endangering further people. Those who are unconscious may require CPR onsite. Administering oxygen via non-rebreather mask shortens the half-life of carbon monoxide from 320 minutes, when breathing normal air, to only 80 minutes. Oxygen hastens the dissociation of carbon monoxide from carboxyhaemoglobin, thus turning it back into haemoglobin. Due to the possible severe effects in the baby, pregnant women are treated with oxygen for longer periods of time than non-pregnant people.

Hyperbaric Oxygen (Reperfusion injury)

Hyperbaric oxygen is also used in the treatment of carbon monoxide poisoning, as it may hasten dissociation of CO

from carboxyhaemoglobin and cytochrome oxidase to a greater extent than normal oxygen. Hyperbaric oxygen at three times atmospheric pressure reduces the half-life of carbon monoxide to 23 (~80/3 minutes) minutes, compared to 80 minutes for oxygen at regular atmospheric pressure. It may also enhance oxygen transport to the tissues by plasma, partially bypassing the normal transfer through haemoglobin. However, it is controversial whether hyperbaric oxygen actually offers any extra benefits over normal high flow oxygen, in terms of increased survival or improved long-term outcomes. There have been randomized controlled trials in which the two treatment options have been compared; of the six performed, four found hyperbaric oxygen improved outcome and two found no benefit for hyperbaric oxygen. Some of these trials have been criticized for apparent flaws in their implementation. A review of all the literature concluded that the role of hyperbaric oxygen is unclear and the available evidence neither confirms nor denies a medically meaningful benefit. The authors suggested a large, well designed, externally audited, multi-centre trial to compare normal oxygen with hyperbaric oxygen. While hyperbaric oxygen therapy is used for severe poisonings, the benefit over standard oxygen delivery is unclear.

Time to remove 50% carboxyhemoglobin

Oxygen pressure O ₂	Time
21% oxygen at normal atmospheric pressure (fresh air)	5 hours 20 min
100% oxygen at normal atmospheric pressure (non-rebreather oxygen mask)	1 hours 20 min
100% hyperbaric oxygen (3 atmospheres absolute)	23 min

Gas Geyser Installation Rules

There are guidelines developed by regulatory agencies for the use of these geysers, including the use of timers, site of installations and dimensions, and ventilation requirements of the room in which they are fitted. As per the Gas installation manual rules all flue less gas appliances have to be labeled with the following wordings: [15]

- 1) Do not operate this appliance before reading the instruction (user guide) booklet.
- 2) Do not place articles on or against this appliance.
- 3) Do not store chemicals or flammable materials, or spray aerosols near this appliance.
- 4) Do not operate with panels, covers or guards removed from this appliance.
- 5) Do not operate in a bathroom or bedroom.
- 6) Do not operate in an unventilated room.
- 7) Do not operate in a room with volume less than (a value dependent on heater capacity).
- 8) Emissions from this space heater may affect persons susceptible to respiratory problems.

2. Discussion

All the cases were brought to the hospital with history of loss of consciousness while bathing. The common link in all the cases was the usage of gas geyser in a small ill-ventilated bathroom. These events occurred in young- to middle-aged individuals without any significant risk factors. Most of the cases were females as they were supposedly exposed for longer duration to the toxic gases due to longer duration of bathing. All the patients regained normal consciousness with

excellent neurological recovery except one case who succumbed to illness. The main composition of LPG is propane and butane (hydrocarbon gases). Other naturally occurring gases such as CO and methane are also present. Sulphur dioxide is added to produce the pungent smell to ensure quick detection of LPG leakage. When adequate oxygen is present, hydrocarbon gases undergo complete combustion to form water and carbon dioxide. However, when oxygen is inadequate, CO and nitrogen oxides (including nitric oxide and nitrogen dioxide) are produced instead. Small amounts of CO are produced due to leakage of LPG tank or from partial combustion of the hydrocarbon gases of LPG. [3] Defective devices, defective installation, and poor ventilation are the frequent causes for the inadequate oxygen required for the complete combustion of hydrocarbon gases. Gas geysers are preferred to electric geysers as it heats up the water faster, cheaper to operate, and no requirement of electricity.

Electric vs. Gas Water Heater	
ELECTRIC	GAS
Cost more to operate.	Cost less to operate.
Last 12 years on average.	Last 13 years on average.
Takes more time to reheat water.	Takes less time to reheat water.
Won't work under power outage.	Will work under power outage.
More efficient. No heat loss.	Less efficient: heat loss.

There are guidelines developed by regulatory agencies for the use of these geysers, including the use of timers, site of installations and dimensions, and ventilation requirements of the room in which they are fitted. CO is a toxic, colorless, and odorless gas, which accumulates rapidly and reaches concentrations, dangerous for humans. It quickly binds to haemoglobin with an affinity 200–250 times higher than that of oxygen to form carboxyhaemoglobin (COHb). Hence, there is a decrease in the arterial oxygen content and shift of the oxyhaemoglobin dissociation curve to the left causing hypoxia. [2] Much of the tissue damage involves inflammatory changes and white cell activation, with increased capillary leakage and oedema. CO has also been shown to impair tissue perfusion through myocardial depression and arrhythmias and peripheral vasodilatation. Acute CO poisoning presents with general malaise, headache, dizziness, and confusion. Coma or seizures can occur in patients with prolonged exposure. [4, 5]

Parts per Million	Time of Exposure	Response
50		Threshold limit, no apparent toxic symptoms
100	Several hours	No symptoms for long periods
200	2-3 hours	Possible headache
400	1-2 hours	Frontal headache and nausea
800	45 minutes	Headache, dizziness and nausea
800	2 hours	Collapse and possible unconsciousness
1600	20 minutes	Headache, dizziness and nausea
1600	2 hours	Collapse, unconsciousness, possible death
3200	5-10 minutes	Headache and dizziness
3200	10-15 minutes	Unconsciousness and possible death
6400	1-2 minutes	Headache and dizziness
6400	0-15 minutes	Unconsciousness and possible death
12800	Immediate	Unconsciousness
12800	1-3 minutes	Danger of death

The classical cherry-red appearance is not seen in all cases of acute poisoning and may not be apparent even in cases of severe toxicity. Symmetrical basal ganglia lesions (figure 5) are one of the radiological abnormalities apparent on MRI. [6]

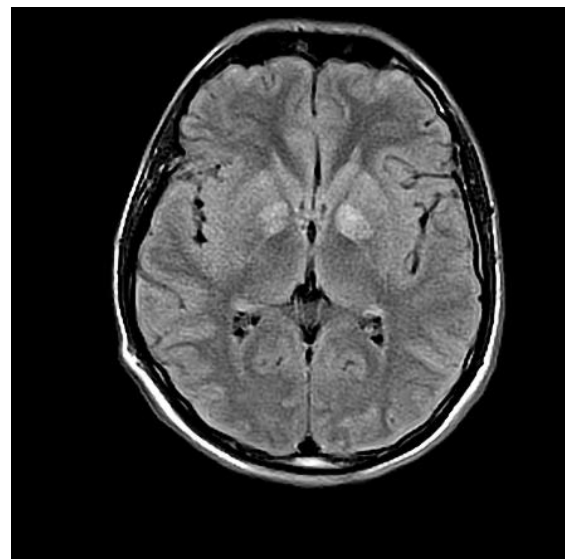


Figure 6: Basal Ganglia Lesions

Hyperoxygenation facilitates the production of partially reduced oxygen species, which oxidizes essential proteins and nucleic acids, causing typical reperfusion injury. CO exposure has been shown to cause lipid peroxidation, leading to reversible demyelination of central nervous system lipids. [7, 8] Immediate death is most likely to be due to a lethal arrhythmia, due to cardiac sensitivity to hypoxia and the preferential binding to cardiac myoglobin. [9]

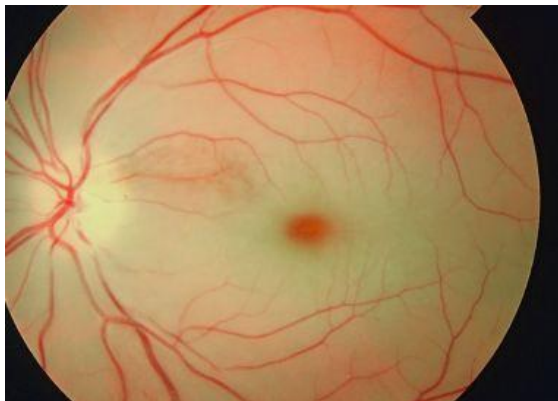
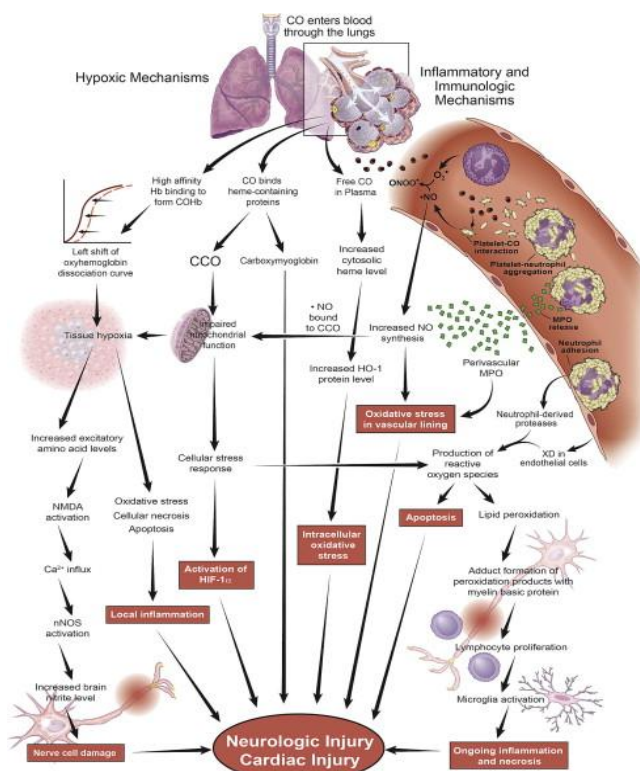


Figure 5: Cherry Red Spot

There are two main types of gas geysers depending on the type of flue (vent) system. One is flueless or room sealed which is the first choice for new installation, and the other is the external flue. In flueless gas geyser, fresh air for combustion and combustion products are taken from and discharged directly to outside air, without contaminating room air. However, flueless gas geyser is considered dangerous as combustion products containing CO, discharge directly into the room, and may build up to dangerous levels. Various studies have shown that flueless gas geyser emits many combustion products that include nitrogen dioxide, CO, and formaldehyde and are often higher than the stipulated guideline levels, especially when the heaters are operated at low gas pressure. [10]



A study in Copenhagen showed that most cases of CO poisoning from gas water heaters were due to sooted convectors and installed without flue. [11] Study by Correia *et al.* on gas geyser syndrome from India reported 26 patients with this syndrome ranging from seizure-like episode to CO intoxication. [1] The effects found in our patients could probably be attributed to LPG combustion

and/or CO intoxication as the levels of COHb levels could not be assessed due to nonavailability. This is the limitation of our study.

3. Conclusion

Awareness regarding CO intoxication due to usage of ill-fitted, ill-ventilated gas geyser is necessary as they are entirely preventable conditions. The clinical features range from mild altered level of consciousness with spontaneous recovery to death. It is also important from medicolegal point of view as most of the patients were female and were found unconscious in the bathroom

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Author Profile



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